DISEASES OF THE HEART
AND CIRCULATION

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DISEASES OF THE HEART AND CIRCULATION

BY

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PHYSICIAN FOR DISEASES OF [illegible]

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TO
MY FATHER

PREFACE TO THE SECOND EDITION

IMPORTANT advances, both in diagnosis and treatment of cardiovascular disease, have rendered a second edition of *Diseases of the Heart and Circulation* desirable in just over four years. Auricular catheterisation has shed new light on the mechanism of cardiac failure; phonocardiography has clarified some of the problems connected with cardiac auscultation, multiple unipolar chest leads have rendered more precise the cardiographic diagnosis of myocardial lesions, and angiocardiography has done the same for congenital heart disease. Knowledge gained by these methods has been incorporated throughout the text. The methods themselves, with the exception of chest-lead cardiography, are essentially matters for the research worker, and no attempt has been made to describe them in detail; but the main principles underlying them have been briefly described, to enable practitioners to recognise the exceptional case in which they may give assistance. For the vast majority of patients, clinical methods with radiology and cardiography give all the information necessary for diagnosis and treatment.

The main plan of the book remains unchanged, but much new matter has been introduced and some sections have been rewritten. The chapters dealing with abnormalities of the ventricular complexes of cardiograms and with congenital heart disease have been largely rewritten, and the latter has been expanded to include additional congenital malformations as well as the surgical treatment of the cyanotic group of cases. The sections dealing with low blood pressure, gallop rhythm, symptomatology of thrombo-angiitis obliterans, and surgical treatment of hypertension have been rewritten. Opportunity has been taken to introduce new material, including sections dealing with the causes, mechanism, symptoms, and treatment of peripheral circulatory failure, the cardiovascular system in various acute infections and in tuberculosis, pneumothorax, temporal arteritis, acute coronary insufficiency, traumatic cardiac lesions, and arterio-venous fistulae. The use of anticoagulants, the penicillin treatment of bacterial endocarditis, the thiocyanate treatment of hypertension, the use of low

PREFACE TO THE FIRST EDITION

In the teaching of medicine I have always endeavoured to relate the clinical aspects of illness to its anatomy, physiology, and pathology. This volume is the outcome of fifteen years' experience in the teaching of medical students, and is based on the notes of lectures delivered at the Anderson College of Medicine. It is thus intended primarily as an introduction to the study of cardiovascular disease, both for students and for practitioners. I have attempted to present the subject in such a way that the reader may be enabled to gain greater insight into his patients and to avoid many of the diagnostic and therapeutic pitfalls which beset him. I have included sections on subjects which are not ordinarily discussed in a course of lectures, but which it is hoped will be of practical value, such as the cardiovascular system in anaemias, the heart in chest deformities, the heart in relation to pregnancy and in relation to surgical operations. No attempt has been made to give a complete bibliography; but in dealing with the less widely known aspects of the subject, and with those sections in which there has been a recent change of outlook, references are given which will serve as an introduction to the literature

I wish to express my grateful thanks to Drs. A. M. Scott and A. Glen, Physicians to the Victoria Infirmary, for permission to refer to their cases and to reproduce the relative illustrations; to Dr. G. J. Wilson, Radiologist to the Victoria Infirmary, for permission to reproduce the teleradiograms; to Dr K. Blum, Assistant Radiologist, for his enthusiastic assistance in the selection of suitable teleradiograms; to Dr. D. M. Harper, Radiologist to Stirling and Falkirk Royal Infirmaries, for his willing help in providing further teleradiograms; to Sister MacPhail of the X-ray Department, Sister Muir of the Outpatient Department, Sisters Stirton and Laird of the Medical Wards of the Victoria Infirmary, for invaluable aid in the collection of material; to Miss Wyper, Technical Assistant

sodium diets in hypertension and in cardiac failure, thiouracil, and theophylline-ethylene-diamine are described and discussed. Eleven new illustrations have been added, three have been omitted, and one has been replaced.

The volume has necessarily been lengthened, but it is hoped that this will prove justified by an increase in its usefulness to students and practitioners.

A. A. F. P.

GLASGOW,
March 1951

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to the staff of the Pathological Department, for the photographs in Figs. 60 A and B; and to many others who have willingly rendered aid. I am also indebted to the Editors and Publishers of the *Glasgow Medical Journal* for permission to reproduce Figs. 28 C, and 53 A to D; and to the Editors and Publishers of the *British Heart Journal* for permission to reproduce Figs. 84 A to C.

A. A. F. P.

GLASGOW,
September 1946

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INTRODUCTION

Disease processes affect the organism in two ways. On the one hand they cause alterations in structure, the study of which is known as *morbid anatomy*. On the other hand they give rise to changes in the behaviour of organs or tissues, that is to say, to changes in physiology; the study of these changes may well be termed *morbid physiology*. It is not customary in the teaching of medicine to deal with morbid physiology as a single subject, indeed the term "morbid physiology" is one which is rarely used, and the student is left to garner such information on the subject as may fall from the lips of diverse teachers—the physiologist, the pathologist, the physician, the surgeon, the biochemist, and many others An attempt is made in the present volume to present the morbid physiology of the circulation side by side with its morbid anatomy, and to show how these determine the symptoms and the physical signs of illness, and form the basis of clinical science

Altered function, broadly speaking, produces various subjective sensations such as pain, headache, breathlessness, nausea, weakness, tiredness, stiffness, etc These subjective sensations are called the *Symptoms* of illness It is in consequence of these symptoms that a patient becomes unable to work, to play, to eat, or perhaps to rise out of bed. It is the symptoms which lead the patient to realise that he is ill; to him an illness consists in the sum-total of the symptoms It is for the relief of these and for removal of the resulting incapacity that he consults a doctor.

Altered structure, broadly speaking, produces signs which are found on examination, such for example as a swelling, a murmur, or an area of dullness on percussion These are called the *Physical Signs* of disease. When teaching medicine in the wards, it is easy to demonstrate physical signs, lumps can be felt, rashes can be seen, murmurs can be heard, and so on. Symptoms, however, usually have to be visualised rather than demonstrated. When a patient says he has a pain or a headache, the doctor can neither prove nor disprove the statement, much less measure the severity of the pain. All he can do is

to get the best description possible from the patient, and try to visualise what the symptom is like. Thus it comes about that in teaching more stress is apt to be laid on physical signs than on symptoms. As a result it sometimes happens that students (or doctors) come to look on illness as the sum-total of the patient's physical signs. The patient's idea of illness and the doctor's may be two entirely different things, sometimes bearing little relationship to one another.

There are some forms of illness which give rise to gross *disturbances of function with little or no alteration in structure.* They produce many symptoms, it may be dramatic symptoms, without any characteristic physical signs. Examples include epilepsy, migraine, paroxysmal tachycardia, some cases of paroxysmal auricular fibrillation, some of spasmodic angina, and many of psychoneurosis. While in many of these conditions the attacks are typical patients may be examined in the intervals between the attacks without any abnormality whatever being found. If the attacks are sufficiently frequent or severe to interfere with the patient's capacity to live a normal life, the patient is ill, despite the negative findings on physical examination. Such illnesses can neither be explained nor understood on a basis of morbid anatomy. their elucidation requires morbid physiology as its starting-point.

On the other hand, there are illnesses which cause structural damage with resulting physical signs. On recovery from the acute stage of such an illness, the structural damage may be repaired, in which case the physical signs disappear. Sometimes, however, a certain amount of permanent structural damage persists for the remainder of the patient's life, this damage may or may not be such as to interfere with the function of the organ affected Provided the disease process has ceased to be active so that the damage is not progressive, *the patient is not ill unless there is interference with function* and resulting disability There are many persons going about who are perfect museums of physical signs yet who are not ill; they feel perfectly well and are able to lead normal lives, working, enjoying themselves, eating, sleeping, and reproducing their kind like their fellow-men. It is of extreme importance *to recognise that physical signs, by themselves, do not constitute illness.* The patient is ill only if the structural damage which the signs represent is causing subjective symptoms and

incapacity, or alternatively, if the structural damage is progressive so that it endangers life and future health.

It is important also that, in those cases in which there are no physical signs, the reality of the symptoms should be recognised. One sometimes hears the suggestion put forward that a particular patient's pain is "imaginary". A few minutes' reflection will show that there is no such thing as "imaginary pain". Pain is a subjective sensation appreciated by the conscious mind. We know that it can be produced by various forms of stimulation of certain sensory nerves. We know that it can also be produced by stimuli arising out of the patient's mental processes By whichever method the sensation has been evoked, it is the same sensation and is equally "real". It is just as absurd to speak of "imaginary pain" as it would be to speak of pain in an unconscious individual. If a patient says he has a pain, he may be deliberately lying, knowing full well that he has no sensation of the sort ; but if he is not lying, his pain is real, and it is the doctor's duty to attempt to elucidate the mechanism whereby it has been produced.

Case taking.—In examining patients, having enquired as to their complaint or complaints, the first step is to take a history. The symptoms of an illness emerge from the history, and a great deal of information about the cause of the illness will often be obtained from the history. A good history is something more than a mere inventory of symptoms with the dates of their development The circumstances in which individual symptoms first appeared should always be noted (whether they may seem relevant at the time or not) ; any change in the patient's mode of life or habits, or any unusual event preceding the onset of a symptom should be recorded. The history is often obtained piecemeal from the patient, but in writing it down, the details should be rearranged in chronological order It is important to get a precise description of symptoms and their behaviour. For this purpose it is usually necessary to cross-examine the patient ; but in so doing care must be taken to avoid suggesting a particular answer to the patient questions should be so worded that no clue is given to the answer expected

The next step is the physical examination. The methods used and the interpretation of the results obtained are discussed in Chapter 3

SECTION I. SYMPTOMS AND PHYSICAL SIGNS IN CARDIOVASCULAR DISEASE

CHAPTER 1

THE NORMAL AND MORBID PHYSIOLOGY OF THE CIRCULATION

THE normal circulation depends on three factors, namely, an efficient cardiac output, healthy vessels, and effective vaso-motor control.

The **cardiac output** in turn requires: (1) an adequate venous return to the right side of the heart; (2) a contraction of the right ventricle sufficiently powerful to supply blood through the pulmonary circulation to the left side of the heart; and (3) a contraction of the left ventricle sufficiently powerful to force blood into the aorta and thence through the arteries to the capillaries under varying conditions of bodily activity and peripheral resistance

Various factors combine to maintain the *venous return*. Firstly, the normal capillaries maintain a constant tone which results in a slightly positive intracapillary pressure. From the capillaries through the veins to the right side of the heart, there is a slight but steady drop in pressure. Secondly, the respiratory movements accentuate this pressure gradient. The expansion of the chest during inspiration produces a negative pressure in the intrathoracic veins, exerting a "suction effect", and drawing blood from the adjacent veins into the venae cavae and right auricle. Thirdly, muscular contractions during exercise "massage" blood along the veins, the valves of which ensure that the flow can only proceed in the appropriate direction There are two main ways in which the venous return [illegible] the first place, loss of fluid or [illegible] pressure gradient, and the [illegible]. This is the mechanism of the *circulatory failure of shock*, and of the *peripheral circulatory failure* which occurs in some severe infections. It is self-evident that if the venous return ceases, the circulation will come to a standstill, however efficient the heart; yet this

is frequently overlooked, and even today these cases are sometimes mis-diagnosed as "heart failure" and treated without benefit by digitalis. The second way in which the venous return may be rendered inadequate is by mechanical obstruction, this occurs in constrictive pericarditis and in cases of pericardial effusion; it is the basis of the congestive failure met with in these conditions, its relief is a mechanical problem.

The cardiac output is closely related to the pressure within the right auricle, which in turn depends on the venous pressure. A rise in right auricular pressure causes an increase in cardiac output. When the output begins to approach the maximum of which the heart is capable, the effect of rising auricular pressure becomes steadily less until cardiac output is maximal. Any further rise in auricular pressure leads to a fall in cardiac output. This mechanism is operative in congestive cardiac failure. It explains the beneficial effect of venesection in suitably selected cases, if the point of maximal cardiac output has been passed, venesection will lower the auricular pressure and allow the output to improve: but if this point has not been reached, a venesection will lower both the auricular pressure and the cardiac output, and will be of no therapeutic value Likewise the effects of intravenous transfusions of blood or saline in cases of anaemia are explained if the point of maximal cardiac output has not been reached, transfusion will increase the output, but if this point has been passed, transfusion, by raising the auricular pressure, will cause a fall in cardiac output and possibly a fatal result

The cardiac output can be determined in man by several methods, that of Cournand and Ranges is most reliable The oxygen absorption per minute is measured as in estimation of the basal metabolic rate. The oxygen content of arterial and venous blood is determined, the latter being obtained by catheterisation of the right auricle (p. 29). Knowing the arterio-venous oxygen difference and the rate of oxygen absorption, the amount of blood passing through the lungs per minute (i.e. the cardiac output) is easily calculated.

The **cardiac efficiency** depends primarily on a strong healthy myocardium; the ventricles are of relatively greater importance than the auricles. Thus, in some patients, the circulation remains comparatively efficient even though the auricles are

not contracting co-ordinately (auricular fibrillation) or are not contracting at the appropriate time (nodal rhythm, heart block) The coronary vessels are essential for the adequate nutrition of the myocardium ; disease of the coronary arteries seriously impairs its efficiency. The quality of the blood is scarcely less important ; anaemia involves an increased circulation rate, thereby throwing additional work on the heart ; at the same time it causes defective nutrition of the myocardium with loss of efficiency.

The chief function of the auricles is to assist in filling the ventricles immediately prior to their systole. During ventricular systole they act as a reservoir to accommodate the inflowing blood, thereby preventing undue venous distension at that time The valves ensure that the propulsive energy of the ventricles is not wasted by forcing blood in the wrong direction, and that no reflux of blood from aorta or pulmonary artery occurs during ventricular diastole . Auricles and valves add to the efficiency of the heart, yet they are less vital than the myocardium , an efficient myocardium can often overcome the disability of an incompetent or stenosed valve and sometimes that of non-functioning auricles, maintaining an adequate circulation despite these handicaps

The Arteries.—By virtue of their elasticity the arteries become stretched during systole to accommodate the extra blood pumped in by the heart. During diastole they recoil, exerting a pressure (diastolic pressure) which maintains the blood flow until the next systole occurs In virtue of their contractility, which is located chiefly in the small arterioles, a resistance is placed in the way of the escape of blood from the arterial system to the capillaries. This resistance, in combination with the diastolic pressure, ensures an even flow of blood to the capillaries instead of an intermittent flow Furthermore, by variations in the peripheral resistance in different parts of the body the flow of blood to different organs can be regulated, one receiving more and another less in accordance with its needs at any given moment.

Arterial disease impairs the efficiency of the circulation. Loss of elasticity causes an increase in pulse pressure, since a greater pressure is required to stretch the rigid arterial wall during systole while the diastolic recoil is less effective and the diastolic pressure falls Extra work is thrown on the left

functions if the arteries are incapable of responding by reason of disease of their walls. The vasomotor system, however, may itself become inefficient, in the absence of any disease of the heart or arteries. This is the basis of the inefficient circulation met with in cases of so-called *effort syndrome*; in these cases the heart and arteries are perfectly healthy, but the circulation is inefficient in consequence of some psychoneurotic state or of some toxic condition. Failure of the vasomotor system is also responsible for *reflex or "primary" shock*.

Rest and Exercise.—Muscle activity is accompanied by dilatation of the arterioles brought about by vasomotor activity and by dilatation of the capillaries brought about chemically. The quantity of blood passing through the muscle and entering the veins is greatly increased, while the muscular contraction accelerates the flow through the veins themselves. The venous return and the venous filling pressure of the right auricle are thus raised. The output of the right ventricle rises (p. 2). This leads to engorgement of the pulmonary circulation followed by an increased venous return to the left auricle, and so to a greater output from the left ventricle. At the same time Bainbridge's cardio-accelerator reflex is evoked; the heart rate is accelerated in consequence of distension of the right auricle.

It will be noted that the output of the right ventricle rises before that of the left ventricle, so that there is a period during which the pulmonary circulation is engorged. At this stage the individual may experience some discomfort and breathlessness. Once the left ventricular output "catches up" he experiences relief, a phenomenon familiar to athletes under the name of "second wind."

The resting circulation rate averages about 5 litres per minute. Healthy persons during moderately strenuous exercise may attain a circulation rate of 12 to 15 litres per minute; while in trained athletes undergoing maximal effort, figures as high as 25 litres per minute have been recorded. Thus, in favourable circumstances, the circulation rate can [illegible]

[illegible] its output is termed the *cardiac reserve*.

ventricle which must now stretch the non-elastic aorta. At the same time the lowered diastolic pressure tends to interfere with the filling of the coronary arteries and to impair the nutrition of the myocardium. Arterial disease also lessens the capacity of the circulation to adjust itself to changes in bodily activity or in posture. Diminished tone of the arterioles leads to a fall in blood pressure and to inadequate nutrition of the tissues, especially the brain. Conversely increased tone leads to a raised blood pressure (both systolic and diastolic) and throws additional work on the left ventricle.

The **capillaries** likewise maintain tone. It is estimated that the total capacity of the capillaries greatly exceeds the total blood volume. In normal circumstances only a small proportion of the capillaries are patent at any given moment, and those which are patent are constantly being changed. Control of the capillaries appears to be chiefly if not entirely chemical. Histamine or a "histamine-like substance" produced in the course of tissue metabolism leads to their relaxation. Substances causing their contraction are so far unknown. It is questionable whether the vasomotor system exerts any direct effect on capillary tone, its action being apparently confined to the arterioles.

In the past the rôle of the **veins** was thought to be largely passive, the flow through them being maintained by the pressure gradient assisted by the respiratory movements and by the effect of contracting muscles; the valves ensure that the flow is in the correct direction Recent work on venous pressure in cardiac failure has led some to believe that the veins themselves have tone, and that they may be capable of influencing the venous pressure, hence the cardiac output, by variations in their calibre It has further been surmised that in certain circumstances they are capable of holding back blood in particular areas such as the splanchnic region.

The Vasomotor System.—The vasomotor system is responsible for co-ordinating the cardiac output and the peripheral resistance with the needs of the body in such a way as to obtain the maximum efficiency with the minimum expenditure of energy. It is also responsible for regulating the quantity of blood supplied to each individual organ or tissue in accordance with its needs at any particular period of time.

It is clear that the vasomotor system cannot carry out its

disease, the capacity for effort is limited by pain rather than by breathlessness (see Angina of effort, pp. 22 and 339).

The mechanism of breathlessness on exertion is not yet entirely clear. Formerly it was attributed to the fact that the circulation rate had not been increased sufficiently to eliminate the products of muscle activity with consequent accumulation of carbon dioxide in the blood and stimulation of the respiratory centre, anoxaemia was also thought to be a factor in severe cases. In cardiac dyspnoea the *venous blood* does in fact contain more carbon dioxide and less oxygen, it is *more acid*. However, this is compensated, in most cases, by the increased pulmonary ventilation, so that the figures for *arterial blood* are usually within normal limits, and sometimes the carbon dioxide content is actually below normal from over-compensation; it is exceptional to find an increased carbon-dioxide content or a diminished oxygen content in the arterial blood. For the majority of cases, therefore, an alternative explanation must be sought, and pulmonary congestion is now thought to be the main factor causing breathlessness. If the cardiac output is not *increased* sufficiently, blood will accumulate in the pulmonary veins in so far as the left ventricle is inefficient, or in the systemic veins in so far as the right ventricle is inefficient. Pulmonary congestion appears to excite a *reflex tachypnoea*; the respirations are rapid and shallow. Although the vital capacity is diminished by *congestion*, the rapidity of the

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The most informative method of gauging a patient's circulatory reserve is by taking a careful history. Enquiry should be made into the way in which he spends his day, and the

Diminished Circulatory or Cardiac Reserve.—An individual may have a circulation which is adequate while he is at rest, but which cannot be increased sufficiently to meet the requirements of exercise, and which therefore becomes inadequate during exertion. This is usually called *diminished cardiac reserve*, though it would be more correct to speak of *diminished circulatory reserve*, as it is often due to some condition other than heart disease. In patients with heart disease it is usually the earliest symptom of the disease.

A diminished circulatory reserve may result from disease of the heart pericardium or arteries, from inefficient vaso-motor control, or from interference with the pulmonary circulation by disease of the lungs. It can also be produced by any general disease which involves an increased cardiac output in the resting state; part of the patient's cardiac reserve is already being utilised at rest, so that a smaller margin remains available for activity. Diseases having this effect on the circulation include thyrotoxicosis and fevers (the basal metabolism being increased), anaemias (in which deficient oxygen-carrying capacity of the blood necessitates an increased rate of flow), and conditions in which there is an abnormal communication between arteries and veins (patent ductus arteriosus, arterio-venous aneurysm, and Paget's disease of bone).

The characteristic feature is that symptoms appear during exertion and pass off soon after the exertion is terminated. In minor degrees, severe effort is necessary to call them forth; as the reserve becomes more diminished, less severe effort will produce symptoms; finally a stage is reached at which the circulation is inadequate even when the patient is at rest, and the condition is now called *circulatory insufficiency* or *circulatory failure* (*cardiac insufficiency, cardiac failure*). The guide to its severity is, not the severity of the symptoms but the amount of effort necessary to produce them.

The chief symptom of diminished reserve is breathlessness on exertion, sometimes accompanied by palpitation or cyanosis. If the exertion is prolonged, oedema may appear (see p. 30). The pulse rate is increased and it remains raised after the cessation of effort until the "lag" in the circulation has been made good. In some patients, particularly those with coronary

disease, the capacity for effort is limited by pain rather than by breathlessness (see Angina of effort, pp. 22 and 339).

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arterial blood. This explanation is in accord with the fact that breathlessness on exertion is usually greatest in those forms of heart disease in which there is considerable pulmonary congestion, and least in those forms in which pulmonary congestion is absent. It has been suggested that distension of the systemic veins is also capable of exciting a reflex tachypnoea. In an individual case, therefore, one of three factors may be responsible for breathlessness—pulmonary congestion, distension of systemic veins, or (occasionally) increased carbon-dioxide content with diminished oxygen content of arterial blood.

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breathing may be taken as evidence that the patient's limit has been reached, provided nervous tachypnoea or sighs are excluded. The pitfalls, however, are numerous, and except in those patients in whom the exercise represents the limit of reserve, the results are of little value.

Compensation.—Heart muscle has great capacity for hypertrophy. If increased work is thrown on any particular chamber the first effect is dilatation of the affected chamber with diminution of the cardiac reserve. Provided the heart muscle is healthy and its blood supply is adequate, the dilatation acts as a stimulus to hypertrophy; the capacity of that chamber becomes increased again, and the cardiac reserve improves once more. A lesion which has resulted in hypertrophy to such extent that the reserve is sufficient to meet all ordinary demands, so that the patient retains an efficient circulation and remains symptom-free, is said to be *fully compensated*, or better *adequately compensated*.

Arterial disease is compensated by the left ventricle, on which the increased strain falls. Valvular obstructive lesions are compensated by hypertrophy of the chamber behind the lesion—aortic stenosis by hypertrophy of the left ventricle; mitral stenosis by hypertrophy of the left auricle, and, if this alone is insufficient, by hypertrophy of the right ventricle; pulmonary stenosis by hypertrophy of the right ventricle, and tricuspid stenosis by hypertrophy of the right auricle. In valvular incompetence (regurgitation), if there is a chamber in front of the lesion, it also takes part in compensation. In the case of aortic regurgitation and pulmonary regurgitation there is no chamber in front of the lesion; compensation is by the left ventricle in the first case and by the right ventricle in the second. In mitral regurgitation the left ventricle and left auricle effect the compensation. During ventricular systole blood regurgitates into the auricle, which also receives its normal quota from the pulmonary veins, and is therefore over-distended; when it empties into the left ventricle, this too becomes over-filled; both the ventricle and the auricle ultimately become hypertrophied. The same process occurs in the case of tricuspid regurgitation, substituting right auricle and right ventricle for left. Disease of the lungs throws additional work on the right ventricle, hypertrophy of which

amount of effort which will cause breathlessness should be noted. Unfortunately, as patients are not all honest in their statements, it is sometimes necessary to use an objective test or *exercise tolerance test.* The scientific way of doing this is to make the patient do exercises of varying severity and duration until his limit is found. Unfortunately this takes too much time for routine clinical use, and instead the patient is often made to do a standard exercise, the effect on his pulse rate and respiration being noted. I cannot emphasise too strongly that the exercise tolerance tests in common use are vastly inferior to the scientific method of testing a patient to his limit, and are also inferior to a good history given by a truthful patient. The tests usually suffer from the defect that the exercise is much too mild and of too short duration to allow any conclusions to be drawn as to capacity for more severe or more sustained effort. The cardiac reserve must be considerably impaired to be detected by some of the tests employed; I have seen them passed with flying colours by patients with gross valvular lesions or even coronary disease. Conversely, simple nervousness may maintain a rapid pulse for longer than usual after the exercise and may lead to the conclusion that the reserve is diminished when it is in fact good. Finally, it does not follow that the response to short exercise of moderate severity gives any information regarding the effect of more sustained effort, in the latter case the phenomenon of "second wind" comes into play I have known a long-distance runner give a very poor response to the short test of running up two flights of stairs, apparently because the exercise was too short to allow him to develop his second wind.

The exercise tests commonly employed include stepping on and off a chair, climbing over steps, climbing stairs, or "toe-touching"; the selected exercise is carried out at a specified rate for a specified time (usually half a minute or one minute) The pulse rate is recorded before the exercise, and afterwards until it returns to its initial level. It is usually stated that this level should be reached in 2 minutes In fact the resting level is often regained in from 30 to 60 seconds; and thereafter the rate may rise again especially in nervous or apprehensive patients. In order to detect the fall, it is necessary to record the number of beats in successive periods of 10 seconds The effect on respiration gives more information Distress in

reach the "arterio-sclerotic age" of 40 to 50 may develop arterial disease or hypertension, and their compensation may break down in consequence. I have seen one patient who developed rheumatic mitral stenosis in childhood, syphilitic aortic regurgitation in her twenties, and who survived several pregnancies and confinements before she finally developed hypertension and died of hypertensive heart failure at the age of 52; the lesions were verified at autopsy

(e) *Acute febrile illnesses*. Fever involves an increased metabolism with an increased circulation rate, and for this reason alone is capable of precipitating a breakdown in compensation. Some fevers produce fresh lesions, for example myocarditis in diphtheria, pericarditis in pneumonia, or endocarditis in tonsillitis Even in the absence of heart disease, febrile illnesses often cause peripheral circulatory failure, and they can equally do so in its presence. Finally, they may leave vasomotor instability, which aggravates any circulatory inadequacy already present from the cardiac lesion

(f) *Toxaemias*. Of these, *thyrotoxicosis* is the most important It is by no means infrequent as a complication of chronic rheumatic valvular lesions. By increasing the circulation rate it may precipitate cardiac failure *Ketosis* is a less frequent cause of a breakdown

(g) *Anaemias* Any anaemia, whatever its type and cause, will impair the nutrition of the myocardium in addition to throwing extra work on the heart, and may therefore precipitate a breakdown in compensation Recognition and treatment of the anaemia restores the nutrition of the heart and restores the pre-existing degree of compensation Diagnosis and correction of the anaemia are just as important as recognition of the original cardiac lesion.

(h) *Hypoglycaemia* involves an increased circulation rate simultaneously with less adequate myocardial nutrition, and affects the heart in a manner similar to anaemia. It is responsible for precipitating failure

the myocardium (for example, myocardial infarction), compensation may be effected by hypertrophy of neighbouring healthy areas of myocardium. Finally, hypertrophy may be diffuse with diffuse lesions, or with combinations of lesions.

Failure of Compensation.—Established compensation may "break down" under various circumstances:

(1) *If the original lesion is progressive.* In some cases of valvular disease, in which there is a true chronic endocarditis, the damage to the valve becomes steadily worse as time goes on. Some cases of hypertension show a steadily rising blood pressure. In these progressive cases there will come a time when compensation has apparently reached its limit; failure appears gradually, and is likely to become steadily worse until it ends fatally.

(2) *If some fresh factor tending to lower the reserve becomes operative.* There are many such factors, one of them, or a combination of several, is responsible for the majority of cases of breakdown in compensation. They include:—

(a) *A fresh cardiac lesion.* A patient whose heart has sustained damage in an earlier attack of acute rheumatism may develop a second attack after an interval; and the second attack often causes additional damage to the heart. Another example is the appearance of subacute bacterial endocarditis in patients with healed valvular lesions or congenital lesions.

(b) *The development of an abnormal cardiac rhythm.* The rhythm which appears is usually auricular fibrillation or auricular flutter. The change in rhythm may be a sequel to an intercurrent acute infection, it may be the result of thyrotoxicosis, or it may arise in consequence of prolonged strain.

(c) *Acute or chronic pulmonary disease.* The breakdown sometimes follows an acute infection of the respiratory tract, either coryza, bronchitis, pneumonia, or pleurisy. The development of chronic pulmonary disease (chronic bronchitis or emphysema) may also precipitate a breakdown.

(d) *Development of arterial disease or hypertension.* Patients with compensated cardiac lesions who

reach the "arteriosclerotic age" of 40 to 50 may develop arterial disease or hypertension, and their compensation may break down in consequence. I have seen one patient who developed rheumatic mitral stenosis in childhood, syphilitic aortic regurgitation in her twenties, and who survived several pregnancies and confinements before she finally developed hypertension and died of hypertensive heart failure at the age of 52; the lesions were verified at autopsy

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(d) *Development of arterial disease or hypertension.* Patients with compensated cardiac lesions who

refuse to give up unsuitable work, or who make a change from a suitable to an unsuitable occupation; they break down in consequence.

(3) *Primary myocardial failure.* Some patients with cardiac lesions remain apparently stationary for years, then develop decompensation in the absence of any of the precipitating factors which have been enumerated. Fishberg regards this as the result of myocardial fatigue, due, in all probability, to inadequate blood supply. Even with healthy coronary arteries, hypertrophy may require a larger blood flow than the vessels can supply. Furthermore, the large myocardial fibres in a hypertrophied heart imply slower diffusion of oxygen and waste products, but many cardiac patients have tachycardia, which allows less time for diffusion, so that nutrition is apt to be inadequate even though the coronary flow is ample In support of this thesis it may be noted that failure is much less frequent in those forms of heart disease which are associated with a slow pulse (e g heart block) than in those which are accompanied by tachycardia.

The recognition of the cause of the breakdown is at least as important as the diagnosis of the cardiac lesion. It depends to a much larger extent on a careful history and general [illegible]

[illegible] and that the heart is failing; but it will give no clue as to why failure appeared at a certain time in a patient whose compensation had previously been good Some of the factors mentioned, by their very nature, imply a progressive failure which is likely to end fatally (for example, a progressive lesion) Others imply failure which is temporary and curable. The patient's reserve will improve to its original state when the precipitating cause of the failure is discovered and successfully treated; in certain circumstances it may be just as important to prescribe iron or liver as to order digitalis. In a third group of cases, namely those in which there has been a fresh cardiac lesion, the symptoms will improve as this lesion in turn becomes compensated, though the patient's reserve is unlikely to reach the same stage of adequacy as it formerly enjoyed.

in some diabetics with cardiovascular disease who *are under treatment with insulin.* Apart from diabetes, hypoglycaemia may arise in myxoedema, in the crises of Addison's disease in pancreatic tumours, and occasionally spontaneously.

(i) *General malnutrition.* If, for any reason, a patient develops malnutrition, the myocardium shares in this, and a breakdown in compensation is likely. Measures directed towards improvement in the patient's general nutrition are likely to have an equally beneficial effect on his compensation

(j) *Psychoneurosis.* Emotional states, particularly anxiety, add the disability of disordered vasomotor control to that of the original cardiac lesion, and they are not infrequent as the precipitating cause of a breakdown in compensation. Many patients *with hypertension experience no symptoms until the* condition is accidentally found at a routine examination (for example, for life insurance, or for entry to the Forces), when they are told about it, they become anxious, and *their circulation, hitherto* efficient, becomes inefficient. The moral is that care should be exercised in deciding whether to tell a patient of such an accidental finding, and in the choice of words used in the event of his being told Prevention is considerably easier than cure in such cases.

(k) *Severe physical strain.* An important example of physical strain causing a breakdown is provided by *pregnancy* and *labour* in women. If compensation of a cardiac lesion is adequate, the strain of pregnancy and labour can usually be borne, on the other hand, if compensation has hitherto been only fair, and certainly if it has been poor, pregnancy is very likely to precipitate failure

The strain of work is frequently blamed for causing a breakdown, but in fact it is a relatively uncommon cause. The majority of patients known to be the subjects of a cardiac lesion are persuaded to take too little exercise rather than too much At least as many deteriorate from insufficient as from excessive exercise. There are, however, a few who

The Mechanism of Peripheral Circulatory Failure or "Shock"

The essential feature of peripheral circulatory failure is a diminution in the circulating blood volume, most often due to loss of fluid, but sometimes to "pooling" of blood in the capillaries or in a localised area of the venous system. The venous return to the heart is lessened and the venous pressure is low, the cardiac output drops, in consequence, and the blood pressure tends to fall. The lowering of blood pressure gives rise, on the one hand to tachycardia by invoking the carotid sinus reflex, and on the other to cerebral anaemia which affects the vasomotor centres producing vaso-constriction. Clinical evidence of vaso-constriction is seen in the pallor of the face and extremities with coldness of the hands and feet. If the venous return is not too greatly lowered, these reflexes may succeed in maintaining the blood pressure at a level of 90 to 100, but in more severe cases they are ineffective, so that the cardiac output and blood pressure fall progressively until death ensues.

The causes of peripheral circulatory failure (also known as "acute hypotension" or "shock") fall into three main groups

(1) *Loss of circulating fluid*

(a) *Haemorrhage* Internal or external. An internal haemorrhage, e.g from a peptic ulcer or from an ectopic pregnancy, produces sudden peripheral circulatory failure which is sometimes mistaken for a "heart attack" The onset of haemorrhagic shock is immediate or rapid. Dilution of the blood after a haemorrhage is a gradual process with steadily falling haemoglobin percentage, when dilution is complete, the picture of shock is replaced by that of anaemia The haemoglobin percentage is therefore unreliable as a diagnostic aid immediately after the onset or as an index of the amount of blood lost during the first two days

(b) *Haemoconcentration* due to *severe diarrhoea* (cholera, dysentery, food poisoning, arsenical poisoning, etc.), *profuse vomiting* (pyloric stenosis, intestinal obstruc-

The Mechanism of Cardiac Failure

In all ordinary forms of heart failure the cardiac output is at first maintained (Harrison, McMichael). In the early stage the venous pressure is raised while the output is still normal. In the late stage of complete incapacity with dyspnoea at rest in bed, the output drops to about 3 litres per minute. McMichael has concluded from these observations that the rise in venous pressure is not the " back pressure effect of a falling output " but rather a compensatory mechanism for the maintenance of optimum output The observed facts are consistent with an alternative, and, to my mind, more satisfying explanation. *Failure develops when the cardiac output cannot be increased sufficiently to meet the requirements of slight effort* During such slight efforts the venous return is increased (p. 5), but the heart, working to its maximum capacity, cannot achieve an output sufficient to prevent a rise in venous pressure. Thus the apparent anomaly of a raised venous pressure and a normal (or even raised) output can be explained on the basis of primary cardiac failure without invoking a hypothetical increase in venous tone. The raised venous pressure does, in fact, tend to maintain the cardiac output at the maximum level of which the heart is capable, and does, therefore, act as a compensatory mechanism, but this fact does not invalidate the argument regarding its mode of production.

Some forms of heart failure are associated with a raised cardiac output (McMichael, Sharpey-Schafer) This occurs in severe anaemias, thyrotoxicosis, beri beri, emphysema, arterio-venous aneurysms, Paget's disease of bone, and congenital cardiac defects which involve a shunt (auricular septal defects, ventricular septal defects, patent ductus arteriosus). They are all conditions which lead to an increased circulation rate before any failure develops, signs of failure appear at a stage when the circulation rate and cardiac output are considerably higher than the average normal The condition has been termed the *hyperkinetic circulatory state* or *hypervolaemia* and the resulting failure is sometimes called *hyperkinetic cardiac failure.* It can arise even with a reduced blood volume such as exists after haemorrhage and in chronic anaemias.

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(b) *Haemoconcentration* due to *severe diarrhoea* (cholera, dysentery, food poisoning, arsenical poisoning, etc.), *profuse vomiting* (pyloric stenosis, intestinal obstruc-

tion, hyperemesis gravidarum, irritant poisoning), or *exudation of plasma* (burns, crush injuries). In cases of burning the exudation of plasma from the raw surface may be sufficient to raise the haemoglobin level to 200 per cent or 300 per cent, implying loss of fluid to the extent of 30 per cent of the total blood volume; analogous haemoconcentration occurs in crush injuries from exudation into the damaged tissues. *Profuse sweating* is seldom the sole cause of shock but may be an added factor in the peripheral circulatory failure of acute infections. The onset of shock due to haemoconcentration is more gradual than that due to haemorrhage; a few hours or a few days may elapse before the symptoms are manifest. Patients with shock due to loss of fluid, whether by haemorrhage or by haemoconcentration, usually have *a dry skin, contrasting with the clammy skin and beads of sweat often seen in other varieties of shock.*

(2) *"Reflex", "neurogenic", or "primary" shock.* This variety of shock is immediate in development and usually occurs in response to *severe pain*. It is found in a wide variety of painful conditions, including coronary occlusion, embolism, rupture of abdominal viscera, biliary or renal colic, and painful injuries. It seems to be due to stimulation of sensory nerves rather than to conscious pain, since it can occur in a lightly anaesthetised patient on handling the peritoneum. At least two distinct varieties of response may follow painful stimulation.

(a) *Vaso-vagal attack.* There is reflex slowing of the heart with transient cutaneous constriction (pallor of face and lips) and simultaneously internal vasodilatation causing a fall in blood pressure. This type of response, which is not a true shock, lasts only a few minutes and ends in complete recovery provided it is not complicated by one of the other varieties of shock. It may occur with minor painful stimuli or from other unpleasant sensations in susceptible persons.

(b) *True primary shock. There is a fall in venous return* with collapsed veins, tachycardia, pallor, and low blood pressure. The mechanism of its production is

not clear, since little is known regarding nervous control of the capillaries; it seems, however, to be due to loss of capillary tone with "pooling" of blood in the capillaries.

(3) *Toxic shock* Many cases formerly attributed to toxaemia with "pooling" of blood in the capillaries are, in fact, due to haemorrhage or haemoconcentration; this is the case in the majority of patients with traumatic shock. Some investigators have discarded the idea of capillary "pooling" entirely, yet there is evidence to incriminate the capillaries as the primary site of the failure in some instances In health the red-cell count of venous and capillary blood differs by less than 3 per cent; in shock, capillary blood may contain 2,000,000 more red cells per c c than venous blood, suggesting that blood stagnates in the capillaries as well as becoming more concentrated there It may be inferred that capillary permeability is increased in these circumstances The conditions in which toxic shock occurs are such as to afford opportunity for toxic damage to the capillaries, thus it often arises in *severe infections*, the toxic agent being bacterial in origin It is found also in certain *metabolic disorders* including diabetic ketosis and the crises of Addison's disease In this connection it may be noted that the suprarenal cortex is often found to be damaged in patients who die from severe infections.

It will be evident that a combination of two or more of the foregoing factors may be operative in a given case of shock Burns and crush injuries may give rise to a reflex primary shock in the first place, followed later by secondary shock due to haemoconcentration, in addition, there is a profound metabolic disturbance and the possibility of capillary damage cannot be entirely excluded Loss of blood and pain may combine to cause the shock of a ruptured tubal pregnancy. In the [illegible]

[illegible] vagal reflex; after some hours the heart rate suddenly drops to about 40 while the blood pressure falls

abruptly and independently of any further reduction in the cardiac output; the fall in blood pressure must therefore be due to vaso-dilatation presumably in the splanchnic area or muscles since the skin remains pale; lowering the patient's head will sometimes abolish this vaso-vagal reflex, restoring the classical picture of shock; in other cases a similar result follows injection of 20 mg. "methedrine" intravenously or 30 mg. intramuscularly.

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CHAPTER 2

THE SYMPTOMATOLOGY OF CARDIOVASCULAR DISEASE

Symptoms in cardiovascular disease, including those related to exertion which have been described as indicating diminished cardiac reserve (p 6) fall into several groups; they may be classified as follows

(1) **Symptoms referable to the heart itself.** Palpitation. Pain.

(2) **Symptoms due to defective circulation:**

- (a) Deficient output into aorta — arterial anaemia
- (b) Venous congestion
  - 1 Pulmonary congestion

  } Left heart failure.

  - 2 Systemic venous congestion
  - 3 Portal congestion

  } Right heart failure.

- (c) Deficient venous return
  - 1 Mechanical obstruction cardiac tamponade.
  - 2 Diminished circulating blood volume } Peripheral circulatory failure or "shock"

(3) **Local disturbance of circulation due to—**

- (a) Embolism
- (b) Thrombosis

(4) **Pressure symptoms.**

These groups of symptoms are not all equally evident in every case, but are determined to some extent by the nature of the heart disease Thus, disease of the coronary arteries is frequently associated with cardiac pain, and is sometimes spoken of as *anginal heart failure*. Hypertension and aortic valvular disease, which increase the strain on the left ventricle, tend to produce the symptoms of arterial anaemia and of pulmonary congestion, particularly in the earlier stages of failure, this variety of heart failure is termed *left heart failure* or *hypertensive heart failure* Mitral stenosis leads to pulmonary congestion followed by systemic and portal congestion, and the condition is spoken of as *congestive*

abruptly and independently of any further reduction in the cardiac output; the fall in blood pressure must therefore be due to vaso-dilatation presumably in the splanchnic area or muscles since the skin remains pale; lowering the patient's head will sometimes abolish this vaso-vagal reflex, restoring the classical picture of shock; in other cases a similar result follows injection of 20 mg. "methedrine" intravenously or 30 mg. intramuscularly.

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failure. Pulmonary disease and right-sided valve lesions cause *systemic and portal congestion, the resulting illness* being called *right heart failure.* Some pericardial lesions obstruct the hepatic vein, causing predominance of portal congestion with a clinical picture so similar to that produced by cirrhosis of the liver that it is termed *pseudo-cirrhotic heart failure.* Constrictive pericarditis and pericardial effusion prevent filling of the heart and give rise to a characteristic variety of failure known as *cardiac tamponade* (*inflow stasis* or *hypodiastolic failure*). Finally, in surgical shock, in haemorrhage, and in some severe infections there is a diminished circulating blood volume, with the result that the venous return to the heart lessens or ceases and the circulation fails in consequence; this is termed *peripheral circulatory failure.*

Many teachers and text-books discuss at length the question as to whether venous congestion is the result of " back pressure " or " *forward pressure* " *The pressure in the circulation* is derived mainly from the heart, and to a slight extent from the respiratory movements ; it acts in the direction —heart—arteries—capillaries—veins—and so back to the heart In the case of a river, if an obstruction is placed in the way of the stream, the level of water rises on the upstream side and falls downstream from the obstruction , the flow still remains in the same direction, though there may be eddies. Similarly, if an obstruction is placed in the way of the circulation, blood accumulates on the " upstream " side of the obstruction , the blood being contained within closed vessels, the pressure rises on this side of the obstruction and falls on the " downstream " side ; but the pressure gradient and flow are still in the normal direction The only exceptions to this rule are, first in the case of valvular regurgitation, and, second, with lesions involving an abnormal communication between the arterial and venous sides of the circulation (atrial or ventricular septal defects, patent ductus arteriosus, or arterio-venous aneurysm) , in these circumstances " back pressure " with a backward flow may occur during systole but during diastole the flow is once more in the normal forward direction. In either event, venous congestion occurs behind (" upstream " from) a lesion, while a fall in pressure occurs in front of it, though the latter is sometimes masked by compensating mechanisms Thus, if the cardiac output is maintained at a normal or nearly normal level, a

reflex vaso-constriction may produce a rise in blood pressure, even during an attack of left ventricular failure.

(1) Symptoms referable to the Heart itself

Heart Consciousness.—The healthy adult is unconscious of his cardiac action. When fatigued, or in circumstances in which the nervous system is hyperexcitable, individuals may become conscious of their cardiac action. This is not a symptom of heart disease, but is merely evidence of an abnormal state of the nervous system whereby the sensory stimuli generated by normal cardiac activity overflow to the paths which arouse conscious sensation.

Palpitation.—"Palpitation" means the conscious perception of forcible or rapid cardiac action. The sensation may be associated with discomfort or slight stabbing pain felt in the region of the cardiac apex. It may be either nervous or cardiac in origin

In nervous persons, and in those suffering from anxiety states, the threshold for conscious sensation is lowered, and normal sensory stimuli from the heart may evoke palpitation. The palpitation is often felt when the patient becomes more excited, or it may be noticed on exertion when the heart rate is increased physiologically.

If the heart-beats are increased in force in consequence of hypertrophy due to disease, palpitation is apt to occur, especially on exertion As long as the cardiac rhythm remains normal, the heart is described as beating regularly during the attack of palpitation

If the heart beats with an abnormal rhythm (whatever the cause and nature) the patient may become aware of the increased rate or increased force of some beats, and he will complain of palpitation. In this case the palpitation is apt to occur in paroxysms irrespective of effort, this occurs with bouts of extrasystoles, paroxysmal tachycardia, and at the onset of auricular fibrillation or auricular flutter. Alternatively the palpitation may be aggravated by effort, as is the case in auricular fibrillation and auricular flutter. The heart may be described as regular or as irregular ("missing beats") during the attack, according to the nature of the abnormal rhythm. An intelligent patient, in describing an attack of palpitation,

can often give a picture sufficiently accurate to permit diagnosis of the rhythm responsible for the attack. As a special form of palpitation under this heading may be cited the consciousness of "missed beats" in patients with extrasystoles or with heart block.

Pain.—Pain can arise in the myocardium or in the central nervous system. There is some difference of opinion as to whether pain can arise in the pericardium or not. It is almost universally accepted that pain cannot and does not arise in the endocardium

Pericardial pain, though denied by some authorities who believe that the pain of pericarditis is really myocardial in origin, almost certainly does exist as a separate entity, distinguishable from myocardial pain. It tends to be felt in the praecordium and to remain strictly localised there though it may on occasion radiate to the left shoulder, it rarely radiates any further It may be associated with hyperaesthesia and tenderness on pressure. It is felt in the early stage of pericarditis when friction is audible, and it tends to disappear as effusion develops. It is not invariably present when friction can be heard, and it rarely recurs when an effusion is disappearing and friction returns, these two points have been cited as evidence against the view that pericardial pain is an entity *distinct from myocardial pain* *It remains constant for some* hours on end, but during that period it may be aggravated by anything which increases the heart rate, for example, by effort It bears many resemblances to pleural pain.

Myocardial Pain.—*Myocardial pain arises when heart* muscle continues to contract in the absence of an adequate blood supply (myocardial ischaemia), and it is analogous to the pain which arises in voluntary muscles which are exercised while their blood supply has been cut off by a tourniquet (MacWilliam's experiment) It therefore occurs in diseases of the coronary vessels, in diseases of the aorta if the orifices of the coronary vessels are narrowed, in aortic regurgitation where the diastolic pressure falls and the filling of the coronary vessels is imperilled, in severe anaemias where the quality rather than the quantity of blood is deficient, and occasionally with paroxysms of very rapid heart action in which the duration of diastole is much reduced. In many of the conditions mentioned, the pain develops during effort when an extra blood

supply is required by the heart, and it passes off shortly after the effort ceases when the extra supply is no longer required (*angina of effort*). In other circumstances the pain develops when a branch of a coronary vessel suddenly becomes obstructed (*angina of decubitus* or *coronary occlusion*). In yet other cases with coronary disease an acute ischaemia may be brought on by factors other than effort, for example by gastric distension, by cold, or by emotion, the resulting attack of pain is known as *paroxysmal* or *spasmodic angina*. These conditions are discussed in greater detail in Chapter 15.

It is sometimes stated that these pains are due to myocardial *anoxaemia*, but this is probably not true, the pains rarely occur in conditions associated with a general anoxaemia such as asphyxia, lung diseases or congestive failure. It is myocardial *ischaemia* which appears to be responsible, in addition to anoxaemia there is deficient removal of waste products

The pain usually starts behind the sternum, often behind the lower or middle third, sometimes behind the upper third. It may remain localised there. Alternatively it may spread across the praecordium or across both sides of the chest; to the shoulders, more often the left, down the inner sides of the arms, especially the left, to the neck or to the lower jaw, again more often on the left side, or to the epigastrium. It is sometimes accompanied by other sensations, for example, a feeling of constriction in the chest, a feeling of suffocation, or a feeling of impending death, but these accessories are not essential to the diagnosis. Rarely, the pain starts in the periphery in one of its areas of radiation, it generally spreads from there to the centre, but in exceptional cases it remains localised at the periphery

Acute distension of one or other of the cardiac cavities is sometimes associated with pain similar to that of myocardial ischaemia. Thus, in cases of pulmonary embolism where there is sudden distension of the right ventricle, there is often pain indistinguishable from that of coronary occlusion. Acute dilatation of the left ventricle (or of the left auricle) with acute pulmonary oedema may likewise be accompanied by anginal pain. While it is possible that distension [illegible]

pain [illegible]

C]

or more cavities are rarely associated with pain. On the other hand, sudden obstruction of the pulmonary circulation by embolism lessens the venous return to the left side of the heart, resulting in a fall in output with consequent diminution in the coronary circulation; left ventricular failure and left auricular failure likewise lead to a fall in output.

Aortic Pain.—The view that anginal pain arises in the aorta as the result of distension was originally put forward by the late Sir Clifford Allbutt, and some physicians still accept this teaching. While it is true that anginal pain occurs in many patients with disease of the aorta, it is the case that the coronary vessels are usually involved in the disease process, so that the pain may nevertheless be myocardial in origin The pain of aortic disease is identical in its situation and behaviour with that of coronary disease. I have not as yet encountered a case of aortic disease in which anginal pain was present when the coronary arteries and their orifices were normal.

Nervous Pain.—In hypersensitive individuals, especially in those suffering from a psychoneurosis and still more in those who are anxious about their hearts, pain may arise in the central nervous system and may be felt in the praecordial area. The mechanism is probably by "overflow" of the normal sensory stimuli from the heart to the "pain tracts". As a rule the resemblance to myocardial pain is only superficial, and the pains can be differentiated by attention to their behaviour. Nervous pain is more often brought on by fatigue or by excitement than by effort; though the patient may say that exertion brings it on, it occurs *after* rather than *during* exertion—when it dawns on the patient that she has done too much, or when she is fatigued—and it is not rapidly relieved by cessation of effort as is true angina of effort; it frequently persists for hours after the effort is terminated. It is more often felt over the apex of the heart than in the mid line, and it may radiate to the left arm, but rarely elsewhere Rarely, it bears a more close resemblance to true myocardial pain Note that a patient can suffer from both types of pain; a person who suffers from coronary disease may be made unduly anxious about his heart from what his doctor or his friends have said.

Referred Pain.—In the diagnosis of praecordial pain it is necessary to remember that pain felt in the praecordium may

be a referred pain due to some other condition; examples include injuries to the muscles or ribs in the praecordial zone, tuberculous disease of the ribs, herpes, spinal disease with root pains at the appropriate level, and pleurisy.

(2) Symptoms due to Defective Circulation

Deficient Output into Aorta.—When the left ventricular output drops, the blood pressure often falls, though, if it was originally very high, it may still remain considerably above normal. Not infrequently, however, the fall in output is accompanied by widespread reflex vaso-constriction; this may be sufficient to prevent any fall in pressure, or even to raise the pressure during the attack. The patients are pale, the lips often appearing quite bloodless in consequence of the vaso-constriction. Sometimes the pallor is accompanied by cyanosis giving rise to an "ashen" or "slaty" colour.

The symptoms are mainly referable to cerebral anaemia, the exact complaints depending on its degree and the rapidity with which it develops. With minor grades there is weakness, light-headedness, dizziness, tinnitus, or faintness. Some patients experience queer sensations; there may be a feeling as though they were floating off the bed into space ("sensation of levitation"); sometimes there is complaint of "pins and needles, starting in the feet and passing up over the body", no doubt due to anaemia affecting the sensory cortex from above downwards. There is breathlessness on exertion brought about by deficient supply of blood to the respiratory centre.

When a more severe grade of cerebral anaemia develops suddenly, as in the *Stokes Adams attacks* of heart block, there is sudden loss of consciousness with extreme pallor and disappearance of the radial pulse. This may be followed by twitchings in the muscles, or by a generalised convulsive seizure resembling epilepsy. The *radial* pulse returns after 30 to 60 seconds, after which the patient regains consciousness and his colour improves. These attacks are especially characteristic of heart block, in which condition they are called "Stokes-Adams attacks", and the *pulse is slow* before and after the seizure; but similar episodes sometimes occur with other disorders of rhythm, for example at the onset of auricular fibrillation, auricular flutter, or paroxysmal tachycardia.

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Referred Pain.—In the diagnosis of praecordial pain it is necessary to remember that pain felt in the praecordium may

cularly will abolish Cheyne-Stokes respiration in many patients. The shoulders should be raised, the legs lowered.

In addition to its effects on respiration, cerebral anaemia of long duration produces mental symptoms. At first there is restlessness and insomnia. Later there is often mental confusion and disorientation, and sometimes delirium. These symptoms, too, tend to become worse during the night. The patients are usually difficult to nurse and difficult to manage; through the night they become noisy, unreasonable, and frequently attempt to get out of bed. The mental state, combined with the Cheyne-Stokes respiration, pallor or pale cyanosis, a fall in blood pressure from its initial level, and symptoms of pulmonary congestion are very characteristic of hypertensive heart failure.

Venous Congestion

Congestion of the Pulmonary Circulation.—Pulmonary congestion occurs early in mitral stenosis, and in cardiac failure secondary to hypertension or to aortic valvular disease. It also develops with any condition causing general heart failure, such as auricular fibrillation or myocardial fibrosis. It may develop suddenly in the form of an attack of *acute pulmonary oedema*, or gradually in the form of *chronic venous congestion of the lungs*

Acute pulmonary oedema is in the majority of cases, due to sudden pulmonary congestion secondary to left ventricular failure or left auricular failure, there are other uncommon causes, such as irritant gas poisoning, acute infections of the lung (pneumonia), and thrombosis of the pulmonary vein The patient is suddenly seized with breathlessness and a sense of suffocation He begins to cough up large quantities of clear froth or yellowish serous fluid, towards the end of the attack the fluid may become tinged with blood and pink in colour. The attack is accompanied by acute distress, and often by collapse, pale cyanosis, and feeble pulse Numerous rales appear all over both lungs; they [illegible]
patient and sufficiently [illegible]
the heart sounds altog[illegible] In some cases the intense pulmonary congestion induces reflex vaso-constriction with a rise in blood pressure in place of the more usual fall, in these

When cerebral anaemia, insufficient in degree to cause loss of consciousness, persists over a long period, as in hypertensive heart failure, it produces a characteristic effect on respiration known as *Cheyne-Stokes breathing*. At first the respirations wax and wane, becoming alternately deeper and shallower. In the more advanced stages the respirations cease altogether for a time, it may be half a minute or a full minute; shallow respirations then appear, and they gradually increase in depth and rate until the patient is severely hyperpnoeic, after a short time they begin to diminish in depth until they again cease. Thus periods of apnoea and periods of hyperpnoea alternate with one another. The condition is thought to be due to diminished sensitivity of the respiratory centre, possibly due to oxygen deficiency, possibly to carbon dioxide deficiency, or possibly to diminished blood flow. A greater concentration of CO_2 is required to stimulate the centre, and the patient has a period of apnoea while the CO_2 is accumulating. Once the centre is stimulated, breathing becomes deep, and the CO_2 is rapidly washed out of the blood, leading to another period of apnoea. A similar type of breathing can result from increased intracranial pressure, whatever the cause of the latter, when intracerebral tension exceeds the arterial pressure, the blood supply to the medullary centres is cut off, causing apnoea; the vasomotor centre, meantime, responds by inducing a rise in arterial pressure, and as soon as this overcomes the intracranial pressure, respiration starts again, but now the blood pressure falls and respiration ceases once more.

Patients with this type of respiration tend to develop it in more marked degree during the night. There is usually pulmonary congestion as well, in which case patients are apt to waken with breathless attacks through the night, this is a very common symptom of hypertensive heart failure. During these attacks the respirations may simply be deep and rapid, but if, as is often the case, there is bronchospasm as well, the hyperpnoea is accompanied by considerable wheezing and the attack is described as *cardiac asthma*. The most effective drug for the relief of these attacks is morphine, which should be given in a dose of $\frac{1}{4}$ grain Although morphine rapidly relieves the respiratory distress it tends to accentuate the periodicity of the breathing. An injection of 0·48 grams of theophylline-ethylene-diamine ("cardophylin") intramus-

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by albuminuria. Distension of the veins in the neck is the result of raised venous pressure. In health the venous pressure at the level of the heart is from 0 to 8 cm. water; in the upright posture the veins in the legs are filled while those above the level of the heart are collapsed owing to the effect of gravity on the venous pressure in different situations. Venous pressure can be directly measured by connecting a wide-bore needle to a vertical manometer similar to that used for spinal manometry; with the patient seated or in the Fowler position and the arm supported so that the front of the elbow is at the level of the right auricle (i.e. 5 cm. below the suprasternal notch), the needle is introduced into the median basilic vein, and any tourniquet is removed; the height to which blood rises in the manometer tube gives the venous pressure. The author inserts a specially designed three-way tap carrying the manometer between the needle and a Record syringe; this permits simultaneous withdrawal of blood or injection of substances for measurement of the circulation time. A rough guide to the venous pressure can be obtained from the level at which distended veins become collapsed or vice versa.

In cases of venous congestion the venous pressure rises so that, even above the level of the heart, a positive pressure exists and the veins are filled; the jugular veins are thus distended. They can often be seen to pulsate. It is important to distinguish between venous pulsation and arterial pulsation in the neck; this is usually easy, even with the unaided eye, for the venous pulse has three waves to each cardiac cycle (two if the auricles are fibrillating) while the arterial pulse has only one. Cyanosis, in cases of systemic venous congestion, is due to slowing of the blood stream in the capillaries; more time is allowed for the reduction of oxy-haemoglobin, leading to an increased proportion of reduced haemoglobin in the venous blood.

Auricular Catheterisation.—Direct measurements of the [illegible] entricle, or pulmonary [illegible] radio-opaque cardiac [illegible] through the median basilic vein until its end lies in the auricle, ventricle, or pulmonary artery; the position of the end of the catheter is determined by fluoroscopy. Pressures are measured by connecting the catheter to a manometer. Alternatively, samples of blood can be withdrawn for determination

circumstances the pulse is bounding rather than feeble and the appearance is livid rather than pale. The attack may prove fatal, but dramatic relief and rapid improvement often follow an injection of $\frac{1}{4}$ grain of morphine, with or without $\frac{1}{100}$ grain of atropine; one or other of these should be given without delay, and the patient should be propped up.

Chronic venous congestion of the lungs gives rise to cough, with a spit which may be blood stained from time to time and in which "heart failure cells" are found microscopically; these are histiocytes which contain altered blood pigment. The patient is breathless. At first the breathlessness is felt only on exertion, but in the more advanced stages of failure it is constant and usually takes the form of *orthopnoea*; by this is meant that the patient is more breathless if he tries to lie flat, with the result that he is usually found propped up in bed with several pillows or leaning over a bed rest. If an orthopnoeic patient slips off his bed rest or pillows during sleep, he is apt to waken with severe respiratory distress, this is another cause of nocturnal breathless attacks in cardiac patients. In the later stages, breathlessness is accompanied by cyanosis due to deficient oxygenation of the blood The chief physical signs of chronic venous congestion of the lungs are rales at the bases of the lungs, often with some rhonchus as well. In early cases fine crepitation may be heard during the first few breaths taken by the patient when he first sits up, after a few deep breaths they disappear In long-standing or advanced cases there is impaired vocal fremitus, impaired resonance and deficient respiratory murmur with rales at the pulmonary bases (the signs of "brown induration of the lung") X-ray shows mottling of the periphery of the lung fields, especially in the lower lobes, and enlargement of the hilar shadows due to distension of the pulmonary veins (*Fig 10*, p. 85); the X-ray picture can readily be mistaken for miliary tuberculosis by the uninitiated.

Other pulmonary signs and symptoms in heart disease may be due to infarction (p. 36), to pleural effusion, or to a complication such as bronchitis or pneumonia.

Congestion of the Systemic Veins.—The symptoms of systemic venous congestion include distension of the veins of the neck; cyanosis; cardiac oedema; hydrothorax; and a fall in urinary output (oliguria) which may be accompanied

factor than congestion of the systemic veins, though the latter also play their part. Cardiac oedema rarely extends above the level of the heart, but if an oedematous patient is propped up, and his hands are allowed to hang at a lower level than his heart, the hands will also become oedematous. If one arm is at a lower level than the other, that hand is more swollen, and the condition can be reversed by turning the patient on the opposite side. Only in extreme cases does oedema extend as high as the neck, if the face is affected, it should raise the suspicion of a coincident nephritic oedema, or of mechanical obstruction of the superior vena cava. In the legs the oedema is symmetrical unless one leg has been supported at a higher level than the other, a discrepancy in the amount of swelling on the two sides suggests venous thrombosis on the more swollen side; the femoral and saphenous veins should be carefully examined.

Venous congestion of the kidneys leads to a diminished output of urine The urine is concentrated, highly coloured, and of high specific gravity; it may contain a trace of albumin, and may show a few red blood cells on microscopic examination; frank haematuria indicates some complication. Congestion in otherwise healthy kidneys does not impair the renal function, and the blood urea remains normal; but congestion affecting previously diseased kidneys (as may be the case in hypertensive patients with congestive failure) is apt to precipitate renal insufficiency with rising blood urea and symptoms of uraemia The combination of symptoms of congestive failure with symptoms of uraemia is sometimes spoken of as *cardio-renal failure*.

Congestion of the Portal Circulation.—Portal congestion occurs under the same conditions as systemic venous congestion, that is as part of congestive or right heart failure. In addition, it can result from obstruction of the hepatic vein by pericardial adhesions or constrictive pericarditis; in these circumstances it may occur without any congestion of the systemic veins, or it may be extreme while systemic congestion is relatively slight. The last mentioned cases provid[illegible] a pictur[illegible]
someti[illegible]
disease [illegible])

One of the chief symptoms is engorgement of the liver (pathologically, a "nutmeg liver") which becomes enlarged

of oxygen content. The method is valuable in the experimental study of heart failure in determination of the cardiac output; it is also helpful in the diagnosis of auricular or ventricular septal defects with left to right shunts, in which circumstances the *oxygen content of auricular (or ventricular blood)* exceeds that of blood from the superior vena cava. For clinical purposes the venous pressure gives the information required for treating patients; in most cases changes in venous pressure run parallel to those in the *right auricular pressure though the figures* are not identical and there are a few exceptions.

Cardiac Oedema.—Oedema means an increase in the amount of fluid in the tissue spaces outside the vascular channels. It may arise from alterations in osmotic balance (nephrosis), from damage to capillary endothelium (inflammation, allergy), or from increase in intracapillary pressure (cardiac oedema). In cardiac oedema, venous congestion leads to increased intracapillary pressure, causing excessive filtration of fluid from the capillaries. This view on cardiac oedema has recently been questioned. Experiments by Brod and Fejfar suggest that cardiac oedema does not depend on changes in venous pressure but on diminished renal blood flow due to vaso-constriction in the *kidney*, with consequent retention of water and salt.

Cardiac oedema appears first in the feet and ankles in ambulant patients, in patients confined to bed it affects the subcutaneous tissue over the lumbo-sacral region first, appearing next round the ankles It then spreads upwards so as to involve the legs, thighs, genital organs, and abdominal wall The oedematous tissues are swollen, pale, and they pit on pressure, on removing the pressure the pit fills up slowly if the pressure of the oedema fluid is low, more rapidly when it is high The looser tissues, such as that over the dorsum of the feet, may be considerably swollen In men the scrotum is sometimes enormously swollen so as to engulf the penis entirely, and the prepuce may also be much swollen; *in women the labia may be similarly affected* An analogous filtration of fluid occurs into the serous cavities. In the pleural cavity this is known as hydrothorax, and it is more common on the right side, though it may occur on either side or on both sides. In the pericardium it is called hydropericardium. In the abdominal cavity it is termed ascites, in this case congestion of the portal veins is a more important

is husky or feeble. Thirst is frequent, especially in cases associated with fluid loss, and in them the skin is dry; in the remainder the skin is clammy and there may be beads of sweat standing out on the face. The skin sometimes shows a bluish-red mottling. Respiration is apt to be shallow and sighing, it may become slow towards the end, especially under morphine.

The pulse is rapid and small, rates of 140 or more being frequent. With the onset of shock the blood pressure falls, the systolic being affected to a greater extent than the diastolic so that the pulse pressure is low. Unless it was enlarged prior to the onset of shock, the heart remains of *normal size*; in fact it probably diminishes in size in consequence of diminished *filling*. Heart sounds may be distant or "tic-tac" (embryocardia), gallop is sometimes heard especially in the shock of coronary occlusion (see pp. 51 to 53 for definition of these signs). The superficial veins are collapsed, *they often fill* inadequately when a tourniquet is applied so that withdrawal of blood is difficult or impossible. Signs of portal congestion are absent.

The urinary output is diminished, in severe cases there is complete anuria. Defective renal circulation leads to impaired nitrogen excretion, with the result that the blood urea rises.

Peripheral circulatory failure is distinguished from left heart failure by the striking absence of *orthopnoea* or signs of *pulmonary congestion*. It is differentiated from right heart failure by the equally striking absence of venous engorgement as well as of hep[illegible]

Treatment.—[illegible] with the head lowere[illegible] [illegible] cause should be treated. *Pain* necessitates morphine in sufficient doses to give relief. $\frac{1}{4}$ grain should be *given immediately* to an adult, and should be repeated in 15 minutes if severe pain persists. In children nepenthe is safer than morphine, the dose being 1 minim for each year of the child's age. Morphine has the advantage of controlling restlessness and allaying anxiety in addition to easing the pain. *Fluid loss* demands replacement by means of an intravenous drip: whole blood is indicated in cases of haemorrhage, plasma for the haemoconcentration of burns or crush injuries, and glucose-saline where fluid loss has resulted from diarrhoea or vomiting. Though theoretically

and is often tender, especially if the enlargement has developed rapidly. The patient may complain of a feeling of weight, or of pain over the liver. Sometimes there is a perihepatitis with pain which is aggravated by breathing or by coughing, and palpable or audible friction. Ascites usually appears soon after the liver congestion has developed, and swelling of the abdomen may be one of the patient's main complaints. It is surprising how frequently the liver function remains adequate despite gross congestion; but in a proportion of cases symptoms of liver insufficiency are present. Almost any of the symptoms of disordered liver function can arise. The most frequent is ketosis which leads to vomiting and causes a characteristic smell of acetone in the breath; jaundice is less common; delirium or coma are very occasional, the blood urea may be raised.

Congestion of the gastric mucosa leads to anorexia, vomiting, flatulence, and epigastric discomfort; occasionally it causes a more or less severe haematemesis. It should be noted that the drug most generally useful in the treatment of cardiac failure, namely digitalis, is a gastric irritant and can cause symptoms very similar to those of gastric congestion. At times it can be difficult to decide whether a patient's gastric symptoms are due to gastric congestion requiring more digitalis, or to the digitalis which has already been given for relief of the congestion. Congestion of the intestinal tract very occasionally causes diarrhoea. Congestion of the spleen produces enlargement of the organ, but it is only rarely that the enlargement is sufficient to render the tip palpable below the costal margin.

Peripheral Circulatory Failure and Shock

The subjects of shock lie flat in bed and are more comfortable without a pillow or if the foot of the bed is raised. Severely shocked patients have a vacant expression and seem oblivious to their surroundings. They appear weak, but may prove surprisingly strong if restraint is required. At first they are apt to be restless, later becoming torpid and stuporose. Sensation is often dulled, some fail to feel pain, becoming conscious of it only when shock passes off. The face is pale, the lips are bloodless, and the extremities are cold. The voice

clot or vegetation is the embolus; and the result is an area of local necrosis in the organ affected known as an infarct, unless there is free anastomotic communication from neighbouring arteries Embolism occurs in malignant endocarditis and subacute bacterial endocarditis where there are brittle vegetations on the valves, but rarely in rheumatic endocarditis, where the warty vegetations are, as a rule, firmly attached to the valve cusps. It occurs in auricular fibrillation, where thrombus frequently forms in the auricular appendages, and sometimes in auricular flutter for the same reason. It may follow coronary thrombosis which sometimes leads to intracardiac thrombus formation It may arise as a sequel to aortic aneurysm from thrombus formed in the sac. Finally, an embolus may originate in systemic veins which are the seat of thrombophlebitis

The embolism may occur at a time when the circulation is otherwise perfectly adequate, this is frequent in bacterial endocarditis Alternatively it may develop as a complication during cardiac failure; this is common in auricular fibrillation. The presence or absence of cardiac failure does, however, influence the consequences of emboli lodging in the lungs; these give rise to infarcts only in the presence of venous congestion

Emboli which lodge in the lungs, producing a lung infarct, come from the systemic veins or from the right side of the heart They occur in auricular fibrillation from thrombus in the right auricular appendix, and in coronary thrombosis from thrombus in the right ventricle. Endocarditis usually affects the valves on the left side, but, when the tricuspid or pulmonic valves are affected lung emboli can result. Endocarditis sometimes affects a congenital lesion such as a septal defect or a patent ductus arteriosus, emboli in such cases are swept into the right side of the heart or the pulmonary artery owing to the higher pressure on the left side or in the aorta; and they consequently lodge in the lungs. Emboli which lodge elsewhere than in the lungs come from the left side of the heart. They occur in auricular fibrillation from thrombus in the left auricular appendix; they are frequent in bacterial endocarditis and not uncommon in coronary thrombosis. The only exception to this rule occurs in the case of patients with auricular or ventricular septal defects, in whom an embolus originating

inferior to whole blood, glucose saline is often effective in haemorrhagic shock; it is of little or no value in burning or crush injuries. *Toxaemic shock* requires treatment appropriate to the particular toxaemia present, e.g. anti-diphtheritic serum, penicillin, sulphonamides, insulin with glucose, etc. Fluids are valuable in these cases also, either by mouth or preferably in the form of an intravenous drip.

An abdominal binder, or an Elastoplast bandage applied to the legs sometimes helps to compensate for the diminished blood volume. Apart from morphine and specific remedies for the various causes of shock, drugs are of doubtful value. Digitalis is devoid of beneficial action and may be directly harmful in that it tends to lower the venous pressure and venous return still further. Nikethamide ("coramine") or "cardiazol" is often given, they are certainly useless in cases due to fluid loss or to toxaemia; they may have some value in reflex cases, but even this is doubtful. "Methedrine" (20 mg. intravenously or 30 mg. intramuscularly) is said to accelerate recovery when shock is complicated by vaso-vagal syncope which does not respond to lowering of the head alone; when vaso-vagal syncope complicates haemorrhagic shock, the value of abolishing the reflex is questionable. Alcohol may have a good psychological effect but is otherwise valueless. Strychnine was formerly popular but there is no evidence that it is effective. Adrenaline is sometimes given, but inasmuch as it acts on the arterioles which are already constricted, it is difficult to see how it can combat the condition. Hopes that suprarenal cortical extract might prove of value have not been borne out by therapeutic experience.

(3) Symptoms due to Local Disturbance of the Circulation

Embolism

In certain forms of heart disease pieces of vegetation from the valves or of clot from intracardiac thrombi are apt to become dislodged; they are carried round in the circulation until they lodge in a vessel which is too small for them to pass through. The process is known as embolism; the piece of

The spleen enlarges, its tip may be palpable, and there is usually tenderness Sometimes friction can be heard over it. It can readily be mistaken for an infarct in the lower lobe of the left lung if the source of embolism is not recognised.

Kidney Infarct.—Sudden pain develops in one or other renal angle, followed by haematuria and possibly by renal colic due to passage of blood clots down the ureter. These symptoms are accompanied by the usual collapse and reactionary fever.

Mesenteric Embolism.—The onset of symptoms is abrupt, with severe abdominal pain, vomiting, and signs of shock, followed by intestinal obstruction. Any faecal matter present in the bowel below the seat of obstruction may be evacuated and blood may be passed simultaneously or independently; but once the lower bowel has been emptied, constipation is absolute. The differentiation from other acute abdominal emergencies depends on the recognition of a possible source of embolism coupled with the fact that the onset of symptoms is more abrupt than with inflammatory lesions.

Limb Emboli.—In most cases there is sudden severe pain in the affected limb, it starts at the point of impaction of the embolus and radiates down the limb Occasionally pain is absent. The pulse is obliterated distal to the point of impaction The limb becomes colder than its fellow. At first it is pale, later blue and cyanotic A collateral circulation may develop subsequently, in which case the colour and temperature of the hand or foot improve; failing development of a collateral circulation, gangrene follows.

Bifurcation of Aorta.—There is simultaneous onset of pain in both legs, with absence of pulse in each and coldness of both feet One leg may be affected to a greater extent than the other. When both common iliac arteries are completely blocked the gangrenous area includes the gluteal region and the lower part of the anterior abdominal wall, the pelvic organs, which derive their blood supply from the internal iliac arteries, also become necrotic. A sufficiently large embolus lodging at the bifurcation of the aorta is capable of blocking the inferior mesenteric artery as well, with consequent gangrene of the descending colon and rectum, in one such case there was gangrene of both legs extending to the brim of the pelvis with incontinence of faeces and passage of blood per rectum

in a systemic vein may pass through the defect to the left side of the heart and cause an embolism elsewhere than in the lungs ("paradoxical embolism").

Wherever it occurs, embolism is characterised by very sudden development of symptoms The onset is often accompanied by shock, with collapse, pallor, and feeble rapid pulse. This is followed by signs of local disturbance of the circulation and frequently by evidence of infarction in the affected organ. If the patient survives the initial shock, there is usually fever and leucocytosis for some days, after which the symptoms gradually improve. *They may disappear entirely, but in some situations the effects are permanent.*

The local symptoms are as follows :

Lung Infarct.—*There is sudden pain in one side of the chest,* often accompanied by breathlessness and followed by haemoptysis. The respirations are rapid, the patient is slaty grey (pale cyanosis) or livid, resembling a pneumonia. Soon after the onset pleural friction is audible if the infarct is superficial, while, later, signs of consolidation appear. X-ray examination will show a circular or wedge-shaped shadow (usually circular); but the shadow is often very transient (Fig. 72, p 349). The fever and other symptoms may last for a few hours, days, or weeks. Blood-staining of the spit usually clears in a day or two but occasionally persists for ten days or more The state of the heart in pulmonary embolism is described in Chapter 16.

Cerebral Embolism.—This is characterised by abrupt loss of function in some part of the brain. The motor area is most frequently involved, so that a sudden monoplegia or hemiplegia appears. Occasionally the visual cortex is affected, giving rise to sudden loss of part of the field of vision Sometimes the embolus lodges in the central artery of the retina, causing sudden blindness in the affected eye I have seen one case of *embolus in the postero-inferior cerebellar artery causing infra-*nuclear facial palsy with dysphagia and crossed anaesthesia (in this particular situation thrombosis is the usual lesion) The shock and reactionary fever so often seen with emboli elsewhere are slight or absent in cases of cerebral embolism

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collapse, followed by fever and leucocytosis for a few days.

are given on the first day, with three of 50 mg. on the second; subsequent dosage is adjusted according to the prothrombin index, bearing in mind that the drop continues for 24 hours or more after the drug has been stopped. A prothrombin index below 20 per cent involves risk of haemorrhage; should this occur, vitamin K is indicated. Heparin affects the clotting-time of the blood, its effect develops rapidly after injection, but is more transient than that of dicoumarin; to maintain the effect, injections must be given every 2 to 4 hours, dosage being controlled by estimation of the clotting-time. Being less toxic, heparin is safer than dicoumarin, but its use involves frequent intravenous injections Neither drug should be used in the absence of facilities for proper control of dosage. If a rapid effect is desired, heparin may be given during the first 2 or 3 days of dicoumarin treatment. (See also note on p. 43.)

Thrombosis

Thrombosis is usually a sequel to arterial disease, especially atheroma, sometimes syphilis It is therefore not uncommon in patients suffering from cardiovascular disease. The type of disease in which it occurs differs from that in which emboli are likely It is most frequent in patients with arteriosclerosis or hypertension, and they are often beyond middle life. In younger patients syphilis may be responsible. Thrombo-angiitis obliterans, temporal arteritis, and periarteritis nodosa are also causes of arterial thrombosis but they are relatively less common Cachectic states, certain blood diseases (e g. leukaemias), and some acute infections (e.g typhoid) tend to precipitate thrombosis Nevertheless thrombosis often occurs in the absence of any obvious precipitating factor Slowing of the blood stream is thought to favour its development in the presence of arterial disease, onset during sleep ...

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during life; autopsy showed necrosis of the descending colon, rectum and uterus, the bladder and the anterior abdominal wall were spared.

Skin Emboli.—Small emboli lodging in the skin vessels produce tender petechiae known as "Osler's spots". They are slightly raised or flat. They appear suddenly, last for a day or two, and while present they are acutely tender. They may occur anywhere, but are especially frequent in the pads of the fingers and toes, and on the thenar and hypothenar eminences. They are most often seen in subacute bacterial endocarditis.

Treatment of Embolism.—The pain in embolism is thought to be due to reflex arterial spasm affecting not only the blocked vessel, but the whole arterial tree in the neighbourhood, the spasm is blamed for much of the circulatory disturbance. In the case of a limb embolism, the spasm and shock should be treated by rest, general warmth, local heat, and injection of ¼ grain morphine or ½ to 1 grain papaverine hydrochloride, the latter drug may be repeated hourly. If the circulation in the hand or foot has not been restored within 6 to 8 hours by this conservative treatment, the operation of embolectomy should be undertaken After 8 to 10 hours the operation becomes impracticable owing to thrombosis in the vessel distal to the embolus and also proximally as far as the next branch. If spasm is not relieved, thrombosis starts even earlier (e g. within two hours) When embolectomy is impracticable, excision of the portion of the vessel containing the embolus may relieve the spasm and restore the circulation of the hand or foot (Leriche). In the case of a large pulmonary embolism, immediate embolectomy offers the best hope of saving life In the case of emboli in inaccessible vessels (e.g in the brain, or within the lung) surgical treatment is impracticable. General medical treatment consists of rest, warmth, and morphine or papaverine to combat shock and reflex spasm.

Anticoagulants.—Anticoagulant drugs (dicoumarin and heparin) are thought to lessen the risk of widespread thrombosis developing as a sequel. Dicoumarin exerts a toxic action on the liver with resulting reduction in the formation of prothrombin. Dosage must be controlled by daily estimation of the prothrombin index which should be maintained at a level between 20 per cent and 30 per cent. Three doses of 100 mg.

are given on the first day, with three of 50 mg. on the second; subsequent dosage is adjusted according to the prothrombin index, bearing in mind that the drop continues for 24 hours or more after the drug has been stopped. A prothrombin index below 20 per cent involves risk of haemorrhage; should this occur, vitamin K is indicated. Heparin affects the clotting-time of the blood, its effect develops rapidly after injection, but is more transient than that of dicoumarin; to maintain the effect, injections must be given every 2 to 4 hours, dosage being controlled by estimation of the clotting-time Being less toxic, heparin is safer than dicoumarin, but its use involves frequent intravenous injections. Neither drug should be used in the absence of facilities for proper control of dosage. If a rapid effect is desired, heparin may be given during the first 2 or 3 days of dicoumarin treatment. (See also note on p 43)

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most common situations for thrombosis are the brain (see Chapter 14), or the coronary vessels (see Chapter 15); the mesenteric arteries and the vessels of the limbs are sometimes affected, though less often.

Treatment.—Arterial thrombosis is rarely if ever amenable to surgical excision. The general medical treatment is similar to that for embolism, viz. rest, warmth, and use of morphine when required for pain or reflex spasm. Anticoagulant drugs may be used in the same manner as for embolism (p 38).

Venous thrombosis. This is comparatively common in persons who have been confined to bed for any reason, but especially where there has been systemic venous congestion, as may be the case in cardiac patients. It tends to occur in the femoral or saphenous veins. It leads to increased oedema on the affected side, and it provides a possible source for subsequent lung embolism. The affected vein is palpable as a firm tender cord if superficial, the adjacent tissue may be swollen and the overlying skin hyperaemic if the thrombosis is a sequel to phlebitis. In patients who are confined to bed for long periods, an attempt should be made to prevent venous thrombosis by gentle massage and passive movements, followed by active movements at the earliest possible moment.

Treatment.—The limb should be kept at rest and elevated, a cage is useful to take the weight of the bed-clothes Local heat, or application of ichthyol and glycerine, eases the pain of phlebitis Anticoagulants are particularly valuable in the treatment of venous thrombosis, they diminish the risk of subsequent lung emboli In some cases the vessel has been ligated proximal to the thrombus with a view to preventing emboli

(4) Pressure Symptoms

Except in the case of aneurysm and pericardial effusion, pressure symptoms are rare in heart disease. A large pericardial effusion causes collapse of the left lower lobe, sometimes of both lower lobes; the resulting physical signs are described in Chapter 7. An aortic aneurysm frequently causes pressure on some or all of the structures in the mediastinum, the symptoms and physical signs are described in Chapter 11. An enlarged left auricle rarely causes pressure symptoms, but dysphagia arising in this way has been described, I have

encountered a case of recurrent laryngeal nerve paralysis in mitral stenosis, presumably from this cause; a few such cases are recorded in the literature.

(5) Summary. The Varieties of Circulatory Failure

Anginal failure is due to disease affecting the coronary vessels. The main feature is anginal pain, viz. angina of effort with or without spasmodic attacks or attacks due to coronary occlusion. Sudden death may occur, alternatively right or left ventricular failure may supervene.

Left ventricular failure may be acute (sudden) or chronic (gradual). Acute left ventricular failure occurs in hypertension, in some cases of coronary disease, and with the onset of abnormal rhythm, especially heart block. There may be an attack of acute pulmonary oedema, or a Stokes-Adams attack, alternatively there is a sudden fall in blood pressure with faintness, dizziness or loss of consciousness, pallor, breathlessness and rapid pulse (except in heart block when the pulse is slow).

Chronic left ventricular failure is characterised by pallor and rapid pulse, dizziness, faintness, or tinnitus progressing to restlessness and insomnia, then confusion or delirium; Cheyne-Stokes respiration, attacks of cardiac asthma, cough and spit with rales at the bases of the lungs followed by pale cyanosis. The rhythm is usually regular but "gallop" or "*triple rhythm*" is common (see p. 53). The blood pressure tends to fall during the course of the illness though at first it may be maintained or even raised.

Acute left auricular failure occurs occasionally in mitral stenosis and takes the form of acute pulmonary oedema with pale cyanosis.

Right ventricular failure (or congestive failure) occurs in its most pure form with heart failure secondary to chronic lung disease or to disease of the pulmonic or tricuspid valve. The symptoms are breathlessness, cyanosis, distension of the neck veins, oedema, oliguria with albuminuria, and portal congestion with a large tender liver and ascites. Auricular fibrillation is common.

In mitral stenosis and in auricular fibrillation congestive failure is accompanied by pulmonary congestion. In addition

to the foregoing symptoms there is orthopnoea, cough, spit which may be blood-stained, and rales at the bases of the lungs.

Pseudo-cirrhotic failure is seen in some cases of chronic pericardial disease where the venous return from the hepatic vein is obstructed mechanically. There are symptoms and signs of portal congestion out of all proportion to other signs of circulatory failure.

Cardiac Tamponade.—The heart itself is diminished in size, though in cases of pericardial effusion this is masked by the presence of the fluid The small heart distinguishes this variety of cardiac failure from right heart failure in which the right side of the heart or the whole heart is enlarged. There is distension of the neck veins with signs of systemic and portal congestion; pulmonary congestion may or may not be present; the pulse is rapid and feeble while the blood pressure is low.

Peripheral Circulatory Failure.—There is pallor with rapid feeble pulse, fall in blood pressure, restlessness or stupor, breathlessness, perhaps sweating, and heart sounds which may be either short and sharp or distant. Absence of distension of the neck veins, of enlargement of the liver, and of pulmonary congestion together with the small size of the heart at once distinguish peripheral circulatory failure from the various forms of heart failure.

The following table from Fishberg's *Heart Failure* summarises the main differences between the chief varieties of circulatory failure:

	Pulmonary veins	Systemic veins	Heart size
Left heart failure	Engorged	Not engorged	Increased
Right heart failure	Not engorged	Engorged	Increased
Tamponade	Variable	Engorged	Diminished
Peripheral failure	Not engorged	Depleted	Diminished

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Note.—Since going to press, several newer anti-coagulant drugs have been on trial. Among others, Tromexan is gaining popularity. Its action is similar to that of Dicoumarin, but more rapid; it is said to be safer, and dosage is more easily controlled. The initial dose should be 150 mg. (half a tablet) six-hourly for four doses, after which the prothrombin time should be estimated.

CHAPTER 3

PHYSICAL SIGNS IN CARDIOVASCULAR DISEASE

Patients who are fit to stand should be examined both erect and recumbent. Current ideas regarding the size and shape of the heart are based on X-ray examination in the erect posture, and it is clearly desirable that clinical assessment of heart size should, when possible, be made under the same conditions. The following description of the cardiac apex and percussion dullness refers to patients examined erect. For auscultation, both positions should be used; some murmurs are audible only when recumbent, while the quality of the heart sounds often improves when erect.

Inspection

Distress in breathing, cyanosis, pallor, distension of the veins in the neck, and oedema, have been discussed in the previous chapter. The position and extent of the cardiac impulse, the presence of visible pulsation elsewhere, and the shape of the chest should be noted

Palpation

The character of the apex impulse is best felt with the flat of the hand, the quality of the normal impulse can only be learnt by practical experience. The position of the cardiac apex should be located with the tip of the forefinger held at right angles to the chest wall Starting in the 5th intercostal space outside the impulse, the finger is moved gradually inwards until a definite thrust from the heart is felt as opposed to transmitted pulsation of the chest wall. The apex is the point furthest downwards and outwards at which this thrust can be felt. It is normally situated in the 5th left intercostal space, 3 to 3½ inches from the mid-line, and within the mid-clavicular line. The impulse covers a rather wider area from ½ to 1 inch in diameter. In broad-chested individuals the apex may be 3¾ or even 4 inches from the mid-line; but in most

cases an apex 4 inches or more from the mid-line is abnormal. The apex moves upwards and outwards on assuming the recumbent posture, it shifts to the right or left if the patient lies on his right or left side. A mobile apex is normal, not pathological.

Abnormal Findings on Inspection and Palpation

Apex Impulse invisible and/or impalpable.—This is often due to a thick chest wall (fat, thick pectoral muscles, or large breasts in women) If the impulse is impalpable with a thin or average chest wall it is an indication of pulmonary emphysema, of pericardial effusion, or of myxoedema.

Diffuse pulsation is often due to excitement and rapid heart action, especially in nervous persons with a thin chest wall. It also occurs in thyrotoxicosis, in acute carditis, in cardiac hypertrophy, and in cardiac dilatation.

A **forcible heaving impulse** is a sign of left ventricular hypertrophy and occurs in hypertension, aortic valvular disease, or with pericardial adhesions. It should be noted that in hypertension, where the left ventricular hypertrophy is concentric, there may be little or no displacement of the apex, in which case hypertrophy can still be recognised from the character of the impulse

A **fixed immobile impulse** is a sign of adherent pericardium

Systolic retraction instead of a systolic thrust is usually a sign of adherent pericardium, but occasionally occurs with extreme right ventricular hypertrophy.

Displacement of the Apex.—(1) A large pleural effusion or a pneumothorax displaces the heart and apex towards the unaffected side (2) Collapse or fibrosis of the lung draws the heart and apex towards the affected side (3) Elevation of the left leaf of the diaphragm (by gastric tympanites, subphrenic abscess, abdominal tumour, ascites, or advanced pregnancy) may displace the apex upwards and outwards to the 4th space 4 or more inches from the mid line On the other hand, eventration of the diaphragm or a diaphragmatic hernia displaces the heart to the right, sometimes to such extent that the impulse lies in the 5th right space and dextrocardia is simulated. (4) The apex is displaced by hypertrophy

of the left ventricle: with the combined hypertrophy and dilatation of aortic regurgitation, the displacement is downwards and outwards to the 6th or 7th space, 4 or more inches from the mid line; with the concentric hypertrophy of aortic stenosis or hypertension the displacement is directly outwards, and the apex remains in the 5th space but lies 4 or more inches from the mid line. In mitral regurgitation the displacement is also directly outwards. (5) In pericardial effusion the impulse is *frequently impalpable*, but *if it can be felt* it may be displaced outwards and upwards to the 4th space.

Pulsation may be seen elsewhere than at the apex:

Neck.—The normal *jugular pulse is exaggerated* in tricuspid incompetence, and abolished in tamponade. *Arterial pulsation* is seen in the suprasternal notch with aneurysm of the arch of the aorta or with chest deformities which displace the arch of the aorta upwards In the side of the neck arterial pulsation may indicate aneurysm of the innominate or carotid artery; but it is frequently seen in thin persons with normal carotid arteries when the pulse pressure is raised, e.g in nervous patients, in thyrotoxicosis, and in aortic regurgitation. In some arteriosclerotic subjects the carotid artery is elongated and tortuous, forming a loop above the clavicle and immediately beneath the skin; this so-called "kinked carotid artery" may be mistaken for an aneurysm, it is more common on the right side Pulsation from enlarged anastomotic arteries is visible in many cases of co-arctation of the aorta, most often in the back (interscapular space) but sometimes in the neck.

Second Interspace.—Pulsation in the second interspace on the right side indicates dilatation or aneurysm of the ascending aorta Pulsation in the second left interspace may be due to a dilated pulmonary artery or to a dilated left auricular appendix.

Epigastrium.—Pulsation in the epigastrium may be due to the abdominal aorta, to the right ventricle, or to an enlarged pulsating liver. *Aortic pulsation* is frequently seen in thin persons; nervous patients sometimes complain of this pulsation. The impulse follows a fraction of a second after the apex impulse, as may easily be demonstrated by placing one forefinger on the epigastric impulse and the other on the apex impulse. Rarely, aortic pulsation in the epigastrium indicates an aneurysm of the abdominal aorta, in this case the pulsating

swelling can be felt and the pulsation is expansile. *Right ventricular pulsation* occurs when there is right ventricular hypertrophy. It is recognised by the fact that the impulse is "inverted", retraction occurring in systole and a thrust in diastole When the heart contracts in systole it becomes smaller, and one would expect retraction to occur in systole with a thrust in diastole; this is, in fact, felt with right ventricular epigastric pulsation The apex thrust occurs in systole because the arch of the aorta tends to uncoil when the pressure in it rises, this throws the apex of the heart forward so that it strikes the chest wall, producing a thrust in systole when retraction might have been expected. *Liver pulsation* occurs in tricuspid incompetence The impulse is either synchronous with the apex impulse or precedes the latter by a fraction of a second, and it is felt more to the right than aortic or right ventricular pulsation

Thrills are best felt with the ulnar border of the hand laid lightly across the chest Th[illegible] of murmurs Sometimes [illegible] detected which is really du[illegible] in the same circumstances as a "split first sound".

Percussion

Percussion of the heart is an art which it has become customary to decry in recent years Admittedly there are certain types of chest in which percussion of the heart is inaccurate or even impossible, such is the case in emphysematous or obese patients, in some women with large breasts, in some men with unusually thick pectoral muscles, and in patients with gastric tympanites. Admittedly, too, percussion is an art which can only be acquired by long practice, and the accuracy attained is largely dependent on the skill of the operator Furthermore, the right border of the heart is always more difficult to define than the left border Having recognised the limitations of percussion, it should be pointed out that there are many chests (in fact the majority) in which percussion by a skilled operator can accurately map out the cardiac borders especially the left border This is the case in thin patients and in those of average nutrition provided they are free from emphysema or gastric tympanites. In such cases

percussion of the left border provides useful confirmation of the information derived from the location of the cardiac apex; and this is especially valuable where diffuse pulsation has rendered precise location of the apex difficult. In cases of pericardial effusion percussion is relatively more easy and more accurate. The student is recommended to practise the art in *all* cases in which the chest is at all suitable, and to check his results by comparison with an X-ray film; at first he will have many disappointments, but a time will come when he can map out on the patient's chest a reasonably close approximation to the left cardiac border as shown on the X-ray.

Percussion should proceed from the resonant towards the dull area, a light stroke gives more accurate results than heavy percussion. In mapping out the *right* border the first point at which a change in note can be detected ("deep" or "relative" cardiac dullness) gives the nearest approach to the radiological outline; but for the *left* border a better correlation with the X-ray appearances is obtained by ignoring the first change in note and proceeding until a definitely dull note is obtained ("superficial" or "absolute" cardiac dullness) If each border is percussed in successive spaces an idea of the cardiac outline can be obtained.

If percussion and palpation have been accurate, the position of the apex as obtained by the two methods should correspond. *From this point the left cardiac border runs upwards* and inwards, forming a curved line, convex outwards, and reaching the left border of the sternum at the level of the third rib. The right border forms a line, slightly convex to the right, reaching its maximum convexity at the level of the fourth right interspace, at which point it may extend about ½ inch from the right sternal border; more often it does not reach beyond the sternal border. The angle between the right border of cardiac dullness and the upper border of liver dullness is acute.

Displaced Cardiac Dullness.—The cardiac dullness is bodily displaced towards the opposite side by a large pleural effusion, pneumothorax, or diaphragmatic hernia. It is displaced towards the diseased side by atelectasis or fibroid lung. Raising of the left leaf of the diaphragm will displace it upwards and slightly outwards. Kyphoscoliotic deformities of the chest will also cause displacement.

Diminished Cardiac Dullness.—The cardiac dullness is diminished by emphysema, and to a less extent by pneumothorax or gastric tympanites. In any of these circumstances, percussion is an inaccurate guide to the size of the heart, though it may provide valuable confirmation of the diagnosis of the lung condition.

Enlarged Cardiac Dullness.—The cardiac dullness is increased to the left by enlargement of the left ventricle, to the right by enlargement of the right auricle, and to both sides by a pericardial effusion. It is not altered by enlargement of the left auricle or of the right ventricle unless extreme, and is therefore of normal extent in uncomplicated mitral stenosis. The *left* auricle enlarges backwards and towards the right; when extreme, this causes extension of cardiac dullness to the *right*. Gross enlargement of the *right* ventricle displaces the left ventricle upwards and outwards, producing extension of cardiac dullness to the *left*; it also involves the pulmonary conus, enlargement of which may cause percussion dullness in the *third left interspace* (Gerhardt's sign). With pericardial effusion the cardiac dullness becomes pear-shaped or conical; the left border is displaced to the left, the upper border is raised to the second rib or higher, and the right border *runs obliquely downwards* and outwards so as to form an obtuse angle with the upper border of liver dullness (Rotch's sign). Extension of dullness either to the *right or to the left* in the second or third interspaces is produced by an aneurysm of the ascending *aorta*. Dilatation of the pulmonary artery may cause percussion dullness in the third, or second and third left interspace.

Auscultation

The normal first sound is a compound sound produced in part by closure of the mitral and tricuspid valves, and in part by the sound of ventricular contraction. *The normal* second sound is produced by closure of the aortic and pulmonic valves. The start of the first sound marks the beginning of ventricular systole. The second sound marks the beginning of ventricular diastole. Events which take place between the start of the first sound and the occurrence of *the second* sound are systolic in time (ventriculo-systolic), events which take place between the second sound and the start of the following first sound are

percussion of the left border provides useful confirmation of the information derived from the location of the cardiac apex; and this is especially valuable where diffuse pulsation has rendered precise location of the apex difficult. In cases of pericardial effusion percussion is relatively more easy and more accurate. The student is recommended to practise the art in all cases in which the chest is at all suitable, and to check his results by comparison with an X-ray film; at first he will have many disappointments, but a time will come when he can map out on the patient's chest a reasonably close approximation to the left cardiac border as shown on the X-ray.

Percussion should proceed from the resonant towards the dull area; a light stroke gives more accurate results than heavy percussion In mapping out the *right* border the first point at which a change in note can be detected ("deep" or "relative" cardiac dullness) gives the nearest approach to the radiological outline, but for the *left* border a better correlation with the X-ray appearances is obtained by ignoring the first change in note and proceeding until a definitely dull note is obtained ("superficial" or "absolute" cardiac dullness). If each border is percussed in successive spaces an idea of the cardiac outline can be obtained.

If percussion and palpation have been accurate, the position of the apex as obtained by the two methods should correspond. From this point the left cardiac border runs upwards and inwards, forming a curved line, convex outwards, and reaching the left border of the sternum at the level of the third rib. The right border forms a line, slightly convex to the right, reaching its maximum convexity at the level of the fourth right interspace, at which point it may extend about $\frac{1}{2}$ inch from the right sternal border, more often it does not reach beyond the sternal border. The angle between the right border of cardiac dullness and the upper border of liver dullness is acute.

Displaced Cardiac Dullness.—The cardiac dullness is bodily displaced towards the opposite side by a large pleural effusion, pneumothorax, or diaphragmatic hernia. It is displaced towards the diseased side by atelectasis or fibroid lung. Raising of the left leaf of the diaphragm will displace it upwards and slightly outwards. Kyphoscoliotic deformities of the chest will also cause displacement.

the ventricles and at the apex (mitral area); in this situation it is a little louder than the second sound. The second sound is best heard at the base of the heart (aortic and pulmonic areas) where it is louder than the first sound. In children the second sound tends to be louder to the left of the sternum than to the right, in adults the second sound is equally loud on each side or is slightly louder on the right side.

When auscultating, the following points should be noted:

(1) The rate and rhythm of the heart These are discussed later (Chapter 5)
(2) The first sound—its intensity, its duration, and whether it is split or not
(3) The second sound—its intensity at the aortic and pulmonic areas, and whether or not it is reduplicated.
(4) The presence or absence of murmurs or friction.

A graphic record of the heart sounds (phonocardiogram) can be obtained with a suitable microphone having the form of the chest-piece of a stethoscope The resulting electric oscillations are amplified and recorded either by a string galvanometer or by a cathode-ray oscillograph, preferably simultaneously with lead 2 of the cardiogram.

The First Sound

The first sound normally has twice or three times the duration of the second sound It is longer when ventricular systole is prolonged, that is in slowly beating hearts whether healthy (athletes) or diseased (myocardial disease, heart block, myxoedema etc.). It [illegible] syst[illegible] ness [illegible] when the heart muscle is weakened, the muscular element in the production of the first sound may be so weak as to render this part of the sound inaudible, the sound is then produced by valve closure only, it is short and sharp, exactly resembling the second sound Sounds of this type are described as *tic tac rhythm* or *embryocardia* because they resemble the normal foetal heart sounds, they are commonly found when the heart is failing as a consequence of a severe febrile illness The first sound is reduced in intensity when there is disease or weakness of the myocardium it also becomes fainter (for reasons which are not clear) when there is delay in conduction between auricles

diastolic in time (ventriculo-diastolic). Events which occur at the end of the diastolic interval, that is, just before the first sound, correspond to auricular systole and are described as "presystolic" or "auriculo-systolic".

Supernumerary sounds.—In some individuals three sounds are heard with each cardiac cycle. The extra sound does not always occupy the same point in the cardiac cycle, and though commonly referred to as the "normal" or "physiological third sound", the fact is that there are third, fourth, and possibly fifth sounds, any one of which may be present in an individual case. Indeed, on rare occasions, four sounds can be heard with each cycle. The *third sound* occurs early in diastole, a fraction of a second after the second sound, giving rise to a rhythm which may be represented by the syllables "lub-te-te". It is frequently heard at the apex in healthy children and young adults, especially when recumbent and resting; it often disappears temporarily if the heart rate changes. It has been attributed to opening of the mitral and tricuspid valves and has been termed "the opening snap of the mitral valve". The *fourth sound* is produced by auricular systole. It occurs later in diastole, synchronising with auricular systole, and being heard immediately before the first sound. It gives the impression of a split first sound, and may be represented by the syllables "t'lub-dup", it is frequently mistaken for a presystolic murmur. It is often associated with a palpable impulse which simulates a bifid apex impulse and may be mistaken for a presystolic thrill. Like the third sound, it is apt to be heard at the apex in healthy adolescents and young adults; but unlike the third sound it is more readily heard in an erect subject. It is by no means uncommon to hear a fourth sound in an erect patient, and a third sound in the same patient when recumbent. Occasionally both can be heard without change of posture. The *fifth sound* is less common and less easily heard. It has been attributed to a sudden rush of blood into the auricle when the latter first dilates after systole, the blood stream striking the closed mitral or tricuspid valve. It therefore occurs during ventricular systole, synchronously with the first sound which usually obscures it; occasionally it can be heard as a short sharp click during the course of the first sound.

In normal circumstances the first sound is best heard over

the second sound may be simulated either by the occurrence of a physiological third sound—in which case it is not pathological—or by a short mid-diastolic murmur; the latter implies mitral endocarditis (early mitral stenosis), and it becomes more obvious in the left lateral position after mild exercise. In arterial hypertension or with a dilated aorta (aneurysm) the second sound becomes loud and ringing at the aortic area (*bruit de tambour*) In pulmonary hypertension, whether primary or secondary, the second sound becomes loud and ringing at the pulmonic area.

Gallop Rhythm (Triple Rhythm)

Gallop rhythm is the term used when there are three sounds of approximately equal intensity with each cardiac cycle; the heart rate is usually increased; the sounds resemble the hoof beats of a galloping horse. From what has been said regarding supernumerary sounds, splitting of the first sound and reduplication of the second sound, it will be seen that there are several varieties of gallop rhythm which differ greatly in their significance.

Protodiastolic gallop is due to first, second, and third sounds. There is difference of opinion as to its significance White found it common in soldiers with neurocirculatory asthenia, and regards it as resulting merely from persistence of a physiological third sound, I have frequently heard this with tachycardia in the absence of heart disease, e g in neuroses, thyrotoxicosis, and fevers Fishberg, on the other hand, believes it to be due to accentuation of the third sound, implying increased intra-auricular pressure, he looks on it as a sign of ventricular failure, left-sided if heard at the apex, right-sided if heard at the lower end of the sternum No doubt the protodiastolic gallop heard in hypertensive heart failure indicates ventricular failure, but that heard in the other conditions mentioned is devoid of diagnostic or prognostic significance. Reduplication of the second sound should not be mistaken for protodiastolic gallop, it is best heard at the base and the two elements of the second sound are closer together than the second and third sound in gallop, the latter is best heard over the ventricles.

Presystolic gallop is produced by first, second, and fourth sounds. A physiological fourth sound usually disappears when

and ventricles (latent heart block). In these circumstances the first sound may become less loud than the second sound at the mitral area, and it is described as a *soft first sound.* It should be noted that, with a thick chest wall or with emphysema, both heart sounds become fainter in equal proportion; the term used to describe this is *distant heart sounds.* Distant heart sounds are often due to extracardiac causes in the absence of any heart disease, or they may be due to pericardial effusion or to thickening of the pericardium; the significance is entirely different from that of a soft first sound.

Sometimes, instead of a single sound, the first sound appears as a double sound or *split first sound* (often inaccurately described as a "reduplicated first sound"). *True splitting of the first sound* occurs if the ventricles are slow in developing the full force of their contraction, so that the sound of valve closure and the sound of muscular contraction become separated by a short interval This is usually due to delay in the spread of excitation through the ventricles, the result of myocardial disease: it is common in chronic coronary disease and in hypertensive heart failure. The splitting of the first sound *may also be due to failure of the mitral and tricuspid valves* to close simultaneously; this sometimes occurs in branch bundle block. In either case, true splitting of the first sound is a sign of myocardial disease, often of serious myocardial disease, it is the basis of systolic gallop rhythm Splitting of the first sound may be *simulated* by a normal fourth sound (see above, p. 50), this is often heard in healthy young adults and is not a sign of heart disease. Secondly, it is possible to mistake a short presystolic murmur for a split first sound (though more often the reverse mistake is made); in this case the true nature of the sound usually becomes obvious when the patient is examined in the left lateral position after mild exercise, a typical presystolic murmur becomes apparent

The Second Sound

Reduplication of the second sound is heard at the base of the heart. It implies that the aortic and pulmonic valves have not closed simultaneously; this may be due to arterial hypertension or to pulmonary hypertension; I am not satisfied that it is necessarily pathological. At the apex, reduplication of

Murmurs

Murmurs are blowing, rustling, or whistling sounds which accompany the normal heart sounds. They are classified, firstly according to the time in the cardiac cycle at which they are heard, and secondly according to the situation in which they are heard and the direction in which they are conducted (if any) According to their timing, they may be Systolic (ventriculo-systolic or v.s), Diastolic (ventriculo-diastolic or v d), or Presystolic (auriculo-systolic or a.s.). They may be heard in the aortic, pulmonic, mitral, or tricuspid area, or elsewhere. Loudness and character have little bearing on their significance ; a loud murmur is more likely to be conducted than a faint one It is important to note whether the heart sounds are heard as well as the murmur, that is, whether the murmur accompanies, obscures, or replaces the sound.

Systolic Murmurs

While systolic murmurs can be caused by valvular defects (stenosis at aortic or pulmonic valves, regurgitation at mitral or tricuspid) there are also many other ways in which they can be produced Some of these mechanisms are understood, others are not Thus dilatation of the left ventricle resulting from hypertension or from aortic valvular disease may lead to dilatation of the mitral ring with regurgitation at the mitral valve , so that a mitral systolic murmur may appear as a sign of aortic valvular disease or of hypertension, the mitral valve itself being healthy Murmurs of this type have been called " secondary " or " irrelevant " murmurs to distinguish them from the " valvular " murmurs due to actual valve damage. In addition to the foregoing, there are many systolic murmurs which occur in the absence of any cardiac disease whatever , they are called " functional " or " innocent " murmurs, sometimes " haemic " murmurs.

Innocent systolic murmurs *Cardiorespiratory murmurs* are usually heard at the apex during inspiration, disappearing during expiration ; occasionally they are heard during expiration and not during inspiration. They are common in healthy adolescents and young adults. The murmur is soft and blowing , it accompanies a first sound of good tone. It disappears if the patient holds his breath at the end of a deep inspiration,

the heart rate increases; occasionally it persists, simulating splitting of the first sound. In presystolic gallop, the sound is loud and distinct; it implies an accentuated fourth sound, and presumably an unusually powerful auricular contraction. It is frequently heard at the apex in cases of hypertensive heart failure; it is sometimes heard at the lower end of the sternum in cases of acute cor pulmonale or of beriberi with right ventricular failure. It implies a grave prognosis, especially in hypertensive heart failure. Ventricular failure is thought to be associated with increased residual blood in the ventricle at the end of systole; a more powerful auricular contraction is therefore needed to overcome the higher ventricular diastolic pressure (Fishberg). This explanation accords with the fact that presystolic gallop disappears when auricular fibrillation develops, and that the extra sound always corresponds to the P wave of the cardiogram, whatever the PR interval, but it is difficult to reconcile with the persistence of presystolic gallop when the auricle fails and pulmonary oedema develops.

Systolic gallop is due to true splitting of the first sound coupled with rapid heart action. It is a sign of serious myocardial disease with a failing heart. It is commonly heard in hypertensive heart failure and in chronic coronary disease. It implies a grave prognosis

Extrasystolic gallop is a rare variety due to the occurrence of alternate sinus beats and extrasystoles. If the extrasystole occurs sufficiently early in diastole it fails to open the aortic valve; it produces a first sound but no second sound, and it sends no pulse wave to the wrist For each pulse wave three sounds are heard, the rhythm being first sound, second sound, first sound, pause It is usually a sign of digitalis poisoning in a heart which has failed to respond to digitalis therapy.

Occasionally in 2–1 heart block the blocked auricular contraction produces a sound which follows the second sound and which may cause a gallop rhythm.

With relatively slow heart rates it is often possible to distinguish between systolic, protodiastolic, and presystolic gallop with the unaided ear. When the heart rate is more rapid, instrumental aid is required. Presystolic gallop can be mistaken for a presystolic murmur. Reduplication of the second sound can be mistaken for protodiastolic gallop.

Dilatation of the mitral ring can be secondary to enlargement of the left ventricle caused by hypertension or by aortic valvular disease, to chronic myocardial disease resulting from coronary sclerosis or from syphilis, to auricular fibrillation, or to myocarditis occurring in diphtheria or in thyrotoxicosis At the tricuspid area, systolic murmurs may be due to analogous conditions affecting the right ventricle and tricuspid ring

Valvular systolic murmurs are accompanied by other physical signs which permit the correct diagnosis to be made ·

(1) *Aortic stenosis.* A harsh systolic murmur is heard to the right of the sternum in the second interspace ; it is conducted to the neck and along the aorta, being audible down the left border of the spine and sometimes in the abdomen. The murmur is accompanied by a thrill and by signs of hypertrophy of the left ventricle It occurs with calcification of the aortic valve cusps as well as in aortic stenosis.

(2) *Aortic endocarditis.* The murmur is often soft and blowing at first, later becoming harsher It is accompanied by signs of ...

... to the left of the sternum in the second and third interspaces ; it is conducted a short distance outwards. There is a thrill with signs of hypertrophy of the right ventricle, cyanosis, and clubbing of the fingers

(4) *Mitral regurgitation* The systolic murmur is usually loud and of high pitch It is heard in the mitral area (i.e. at the apex) and is often conducted towards the left axilla or to the angle of the left scapula It tends to replace rather than accompany the first sound There are signs of enlargement of the left ventricle, often with a thrill If accompanied by a presystolic or mid-diastolic murmur, that is, by signs of mitral stenosis, the diagnosis is justified Mitral regurgitation without stenosis is rare If signs of mitral stenosis are absent a diagnosis of organic mitral regurgitation is only justified if all secondary murmurs have been excluded, especially those of hypertension or aortic valvular disease A rheumatic history is valuable as confirmation though not essential. Many physicians refuse to recognise a diagnosis of mitral regurgitation. According to Evans the systolic murmur of

sometimes at the end of a full expiration. It often disappears in the erect posture. *Tachycardia:* in rapidly beating hearts whether due to excitement, fever, thyrotoxicosis, or other cause, a soft blowing murmur often accompanies a loud slapping first sound at the mitral area. In the absence of any other signs of heart disease the murmur may be disregarded. *Anaemia* frequently gives rise to a blowing systolic murmur ("haemic murmur"), loud or faint, and heard either at the base, at the apex, or both; it disappears with cure of the anaemia. *Posture* systolic murmurs which may be loud or soft are sometimes heard with patients recumbent, disappearing when they are examined erect. A systolic murmur *is occasionally heard in the pulmonic area when the pretracheal fascia* is put on the stretch by bending the head backwards (Eustace Smith murmur); it disappears on flexing the head. It is to be noted that the foregoing murmurs occur in persons who show no other evidence of heart disease, and in these circumstances they may be entirely disregarded. Occasionally a systolic murmur which may be loud or soft and which fails to conform to any of the foregoing murmurs is heard at the apex if it is unaccompanied by any other evidence of heart disease after careful and full examination, it may be disregarded. Evans claims that innocent and secondary can be distinguished from valvular systolic murmurs in phonocardiograms, since valvular murmurs start at or before the S of the cardiogram while innocent and secondary murmurs start after S This claim has been questioned by other workers. It is a fact that, clinically, the first sound can sometimes be heard before a systolic murmur, the latter occupying the interval between first and second sounds These *late systolic murmurs* may be innocent or secondary, but are never organic valvular murmurs.

Irrelevant or secondary systolic murmurs These are also very common Systolic murmurs at the aortic area may be due to roughening of the aortic intima by atheroma or by syphilis, to dilatation of the aorta in syphilis or in hypertension, or to anaemia At the pulmonic area they may be caused by anaemia, or by dilatation of the pulmonary artery secondary to mitral stenosis, to emphysema, or to other chronic lung disease. At the mitral area they are due to dilatation of the mitral ring, which may be either temporary or permanent.

Mitral regurgitation should not be diagnosed unless all the criteria specified above have been fulfilled.

Diastolic Murmurs

In marked contrast to systolic murmurs, diastolic murmurs are almost invariably organic. Most physicians state that they are always organic. This is very nearly true, but not quite; on rare occasions a diastolic murmur is "irrelevant", and on still more rare occasions it is transient and apparently "innocent". For practical purposes, however, a diastolic murmur should always be deemed organic until the contrary has been proved beyond any shadow of doubt, even when there are no other signs of heart disease.

Diastolic murmurs are sometimes subdivided according to the time in diastole at which they occur; thus a murmur may be described as early, mid, late, or full diastolic. Early diastolic murmurs either replace the second sound or follow it without any pause; they arise in the aortic or pulmonic valve. Mid-diastolic murmurs arise in the mitral or tricuspid valve; they are separated from the second sound by a short interval, the start of the murmur corresponding in time to the third heart sound. Late diastolic murmurs are also produced at the mitral or tricuspid valve; they correspond in time to the fourth heart sound (auricular systole), beginning later in diastole and continuing until the succeeding first sound, provided there is no delay in auriculo-ventricular conduction; they are presystolic in time and will be discussed under that heading. Full diastolic murmurs usually arise in the aortic or pulmonic valve; and they generally imply a later stage of illness or a greater degree of damage than early diastolic murmurs. The defect indicated by a diastolic murmur at the aortic or pulmonic area is regurgitation, at the mitral or tricuspid area, stenosis (provided the murmur is not conducted from the aortic area).

(1) *Aortic regurgitation.* The diastolic murmur, which is often soft though sometimes loud, is heard in the 2nd right intercostal space; it is conducted downwards and to the left, along the left border of the sternum to its lower end, and in some cases to the mitral area. The murmur may occupy the early part of diastole only, or the whole of diastole. If faint, it is heard only along the left border of the sternum or only at

mitral regurgitation starts during auricular systole and is indistinguishable in phonocardiograms from the presystolic murmur of mitral stenosis; be this as it may, the two murmurs sound entirely different.

(5) *Mitral endocarditis.* When it first appears the murmur is a soft murmur accompanying a soft first sound, and it may be impossible to distinguish from an innocent or an irrelevant murmur. The circumstances in which it appears are important, viz. during the course of acute rheumatic fever or an illness suggesting acute endocarditis. *Prolonged observation with a careful watch for concomitant signs may be required, and a positive diagnosis of endocarditis is not justified until some other sign or signs have appeared.*

(6) *Tricuspid regurgitation.* A systolic murmur is heard just to the left of the lower end of the sternum. It is accompanied by signs of enlargement of the right auricle, by an enlarged pulsating liver, and by distended pulsating neck veins. Organic tricuspid regurgitation is rare while secondary tricuspid murmurs are common. The diagnosis should not be made unless the concomitant signs are present or signs of tricuspid stenosis coexist.

(7) *Ventricular septal defect* There is a thrill with a systolic murmur, audible over a wide area but maximal at the junction of the fourth left costal cartilage with the sternum. It is loud, usually replacing the first sound, and it is uninfluenced by exercise, posture, or respiration. The heart may be enlarged, but often the murmur and thrill are the only clinical signs, X-ray being necessary to demonstrate the altered shape of the heart shadow, which becomes more globular. *Atrial septal defect* sometimes produces a similar murmur with its point of maximal intensity at a slightly higher level With either of these defects there is occasionally a diastolic murmur as well as a systolic.

It should be noted that from 50 to 75 per cent of all systolic murmurs are due to some cause other than organic valvular disease. A systolic murmur should be considered an indication for complete and careful cardiovascular examination. If this fails to show any other evidence of heart disease, the murmur should, without hesitation, be pronounced innocent, and it should thereafter be ignored. A systolic murmur as an isolated finding should never be taken as evidence of heart disease.

Mitral regurgitation should not be diagnosed unless all the criteria specified above have been fulfilled.

Diastolic Murmurs

In marked contrast to systolic murmurs, diastolic murmurs are almost invariably organic. Most physicians state that they are always organic. This is very nearly true, but not quite; on rare occasions a diastolic murmur is "irrelevant", and on still more rare occasions it is transient and apparently "innocent". For practical purposes, however, a diastolic murmur should always be deemed organic until the contrary has been proved beyond any shadow of doubt, even when there are no other signs of heart disease.

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(1) *Aortic regurgitation.* The diastolic murmur, which is often soft though sometimes loud, is heard in the 2nd right intercostal space, it is conducted downwards and to the left, along the left border of the sternum to its lower end, and in some cases to the mitral area. The murmur may occupy the early part of diastole only, or the whole of diastole. If faint, it is heard only along the left border of the sternum or only at

the lower end of the sternum. It is usually more easily heard if the patient is examined sitting up and leaning slightly forward, or *lying on his left side*. *Diastolic murmurs* are more difficult to detect than systolic murmurs of like intensity; patients are frequently referred for an opinion on a mitral systolic murmur (irrelevant) in whom an aortic diastolic murmur has been overlooked. I would particularly emphasise that the diastolic murmur is the *earliest sign* of aortic regurgitation, and that the diagnosis should be made on the murmur alone in the absence of any other abnormal findings. Other signs of aortic regurgitation which appear later are a heaving impulse with downward and outward displacement of the apex, a water-hammer pulse with high pulse pressure and capillary pulsation, and *carotid pulsation in the neck*. In the early stages patients are of normal colour; in the later stages they are apt to be pale.

(2) *Pulmonary regurgitation.* Organic pulmonary regurgitation *due to endocarditis is rare. Less infrequent, though* still uncommon, is a *functional* pulmonary regurgitation secondary *to dilatation of the pulmonary artery*; this may be secondary to chronic lung disease or to mitral stenosis. The murmur *is similar to that of aortic regurgitation* but is heard down the left border of the sternum only. The concomitant signs are different. There is cyanosis in place of pallor, and signs of right ventricular enlargement in place of left. The pulmonary diastolic murmur which is sometimes heard in mitral stenosis due to dilatation of the pulmonary artery is known as the Graham Steell murmur; it is an example of an "irrelevant" diastolic murmur.

(3) *Diastolic murmurs at the mitral area* are either conducted from the aortic area or else indicate mitral stenosis. A full diastolic murmur is more likely to be a conducted aortic murmur, while a mid-diastolic murmur is more likely to be of mitral origin. Conducted murmurs can usually be traced quite easily back to their point of origin. The mid-diastolic murmur of mitral origin indicates the second stage of mitral endocarditis (that is, the earliest stage of mitral stenosis); in the later stage it is replaced or accompanied by a presystolic murmur. The murmur alone, without any other physical signs, justifies the diagnosis of mitral stenosis. Care is required not to confuse it with a physiological third sound.

(4) *Diastolic murmurs at the lower end of the sternum* are in most cases conducted from the aortic area, occasionally from the pulmonic area. In a small number of cases they indicate tricuspid stenosis, in which case the murmur is usually mid-diastolic, and it may be replaced by a presystolic murmur subsequently.

(5) Very rarely a diastolic murmur heard at the left edge of the sternum is a sign of an atrial or ventricular septal defect.

Presystolic Murmurs

Like diastolic murmurs (of which they are really a variety), presystolic murmurs are almost invariably organic. Most physicians believe them to be organic in every case. Again this is not strictly accurate. There is one presystolic murmur, which is not due to disease of the valve in whose area it is heard, but is secondary, namely the "Austin Flint murmur" of aortic regurgitation, heard in the mitral area. Crighton Bramwell (in company with certain American authors) describes a *functional mitral presystolic murmur*. According to Bramwell, a presystolic murmur can be produced, either by narrowing of the mitral valve, or by an increased rate of flow through a valve of normal diameter. Though extremely rare, I have seen this on two occasions. Both patients were severely anaemic, both had typical mitral presystolic murmurs which led me to diagnose mitral stenosis without any hesitation; in neither case was there any radiological evidence of enlargement of the left auricle, and in both cases the murmur disappeared entirely when the anaemia was corrected. Theoretically it is possible to visualise also a *relative mitral stenosis* if the mitral ring remains of normal size while left auricle and left ventricle both become dilated. I have seen one case in which this seems to have happened, in which a presystolic murmur appeared after a coronary thrombosis which led to aneurysm of the left ventricle.

For practical purposes, however, presystolic murmurs are almost invariably organic, and should be assumed to be organic until proved otherwise. At the mitral or tricuspid area they indicate stenosis. The murmur may precede any other abnormal finding; it may precede X-ray or cardiographic change; consequently a diagnosis may be made on the murmur alone with a 99 per cent probability of its being correct.

the lower end of the sternum. It is usually more easily heard if the patient is examined sitting up and leaning slightly forward, or lying on his left side. Diastolic murmurs are more difficult to detect than systolic murmurs of like intensity, patients are frequently referred for an opinion on a mitral systolic murmur (irrelevant) in whom an aortic diastolic murmur has been overlooked. I would particularly emphasise that the diastolic murmur is the *earliest sign* of aortic regurgitation, and that the diagnosis should be made on the murmur alone in the absence of any other abnormal findings. Other signs of aortic regurgitation which appear later are a heaving impulse with downward and outward displacement of the apex, a water-hammer pulse with high pulse pressure and capillary pulsation, and carotid pulsation in the neck. In the early stages patients are of normal colour, in the later stages they are apt to be pale

(2) *Pulmonary regurgitation.* Organic pulmonary regurgitation due to endocarditis is rare. Less infrequent, though still uncommon, is a functional pulmonary regurgitation secondary to dilatation of the pulmonary artery, this may be secondary to chronic lung disease or to mitral stenosis. The murmur is similar to that of aortic regurgitation but is heard down the left border of the sternum only. The concomitant signs are different. There is cyanosis in place of pallor, and signs of right ventricular enlargement in place of left. The pulmonary diastolic murmur which is sometimes heard in mitral stenosis due to dilatation of the pulmonary artery is known as the Graham Steell murmur; it is an example of an "irrelevant" diastolic murmur.

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septal cusp of the mitral valve while a second stream of blood from the auricle impinges on its auricular surface.

Continuous Murmurs

In certain circumstances murmurs continue right through the cardiac cycle; they may wax and wane, becoming louder in systole and fainter in diastole, but they do not cease entirely. They must not be mistaken for double murmurs, that is to say for separate systolic and diastolic murmurs in the same area; these merely have the combined significance of the individual murmurs. A continuous murmur indicates an abnormal communication between the arterial and venous sides of the circulation. A *patent ductus arteriosus* produces a continuous murmur, louder in systole and fainter in diastole, heard in the 2nd left intercostal space about an inch from the left sternal border (Gibson murmur). *Arterio-venous aneurysms* produce a similar murmur heard over the aneurysm, though they do occur occasionally in the chest, they are uncommon there, they may be found in the limbs, or in the skull. *Atrial or ventricular septal defects* occasionally produce a continuous murmur, but more often the murmur is systolic only.

Venous hum. In certain patients a faint continuous "humming" sound is heard throughout systole and diastole. It is known as a venous hum, and is of no significance. It can sometimes be heard over the jugular bulb, and occasionally in the chest.

Pericardial Friction

Pericardial friction is a sign of pericarditis. It may be heard over any part of the praecordial area, but usually it appears first and persists for longest at the base of the heart. It is generally easy to distinguish from a murmur, being altogether coarser and harsher, it may resemble the sound produced by sawing wood, by sandpapering, or by creaking leather. Sometimes it is a softer sound more closely resembling a murmur; but the systolic and diastolic elements of friction frequently fail to synchronise exactly with systole and diastole as gauged by the heart sounds. The systolic element may start a little after the first sound, ending a little after the second sound, while the diastolic element may overlap the

Presystolic murmurs are frequently diagnosed when no such condition is present. Common mistakes are to regard a split first sound as a presystolic murmur, or to label a loud or harsh systolic murmur "presystolic". A true presystolic murmur is very characteristic, and a murmur should never be labelled presystolic unless it is typical. The murmur starts faintly at about mid-diastole and it runs up to a loud slapping first sound in which it ends; to the ear, it has a crescendo quality, though Bramwell maintains that this is an illusion due to the slapping first sound which follows. In any case of doubt, the characteristic features of the murmur can usually be brought out by exercising the patient and then examining him in the left lateral position.

Mitral stenosis. The presystolic murmur is heard just internal to the apex. It may be the only sign of mitral stenosis, and while the shape of the heart shadow is usually characteristic, the presystolic murmur may precede this change. In uncomplicated mitral stenosis the apex is not displaced. Signs which develop later are a presystolic thrill, accentuation of the second pulmonic sound, and right ventricular pulsation in the epigastrium. Other murmurs which may be present are a mid-diastolic mitral murmur, a systolic murmur due to concomitant mitral regurgitation, or a Graham Steell murmur due to pulmonary regurgitation. The presystolic murmur disappears if the rhythm changes, that is, if auricular fibrillation, auricular flutter, or nodal rhythm supervene; if latent block develops, the murmur becomes mid or early diastolic.

Tricuspid stenosis. The presystolic murmur is heard at the left border of the lower end of the sternum. In character it resembles the murmur of mitral stenosis, and it disappears in the same circumstances. Other findings are thrill, signs of enlargement of the right auricle, and enlargement of the liver; there is striking absence of orthopnoea in the presence of signs of cardiac failure which would ordinarily be associated with severe orthopnoea.

Austin Flint murmur. This is a secondary presystolic murmur, heard at the mitral area in cases of aortic regurgitation who have no mitral stenosis. It is not very common; in most cases of aortic regurgitation with a mitral presystolic murmur, mitral stenosis is present. The Austin Flint murmur is thought to be due to a regurgitant stream of blood impinging on the

septal cusp of the mitral valve while a second stream of blood from the auricle impinges on its auricular surface.

Continuous Murmurs

In certain circumstances murmurs continue right through the cardiac cycle, they may wax and wane, becoming louder in systole and fainter in diastole, but they do not cease entirely. They must not be mistaken for double murmurs, that is to say for separate systolic and diastolic murmurs in the same area; these merely have the combined significance of the individual murmurs. A continuous murmur indicates an abnormal communication between the arterial and venous sides of the circulation. A *patent ductus arteriosus* produces a continuous murmur, louder in systole and fainter in diastole, heard in the 2nd left intercostal space about an inch from the left sternal border (Gibson murmur). *Arterio-venous aneurysms* produce a similar murmur heard over the aneurysm, though they do occur occasionally in the chest, they are uncommon there, they may be found in the limbs, or in the skull. *Atrial or ventricular septal defects* occasionally produce a continuous murmur, but more often the murmur is systolic only.

Venous hum In certain patients a faint continuous "humming" sound is heard throughout systole and diastole. It is known as a venous hum, and is of no significance It can sometimes be heard over the jugular bulb, and occasionally in the chest

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pressure takes place on inflating the armlet, in these cases it will be noted that the pulse and sounds disappear at a certain point during inflation, the inflation is stopped above this level, but before deflation can be commenced, the sounds "catch up" and become audible again; the whole process may be repeated on further inflation In such circumstances it is advisable to record the blood pressure obtained on inflation instead of on deflation of the armlet, patients reacting in this way are usually nervous and are often found to give a normal response at a subsequent date

The method agreed by a Joint Committee representing the Cardiac Societies of Great Britain and America is as follows: The sphygmomanometer cuff is applied above the elbow, avoiding folds The cuff is inflated until the radial pulse disappears A stethoscope is then applied in front of the bend of the elbow, and the pressure is released in the cuff, allowing the mercury to drop about 5 mm at a time The point at which beats are first heard is the systolic pressure. As the pressure is further released there are successive changes in the sound heard, there may or may not be a silent interval following the sharp sound first heard, then the sound becomes a murmur next it becomes [illegible]

the [illegible]

poir[illegible]

metres lower, the dull sound becomes inaudible (5th point). The procedure should be repeated after fully deflating the cuff If the second reading differs appreciably from the first a third reading should be taken, and so on until a constant value is obtained The Societies recommend that the pressure should be recorded to the nearest 5 millimetres, as variations of less than this are devoid of significance Many physicians, nevertheless continue to record blood pressure to the nearest 2 millimetres.

Normal Blood Pressure.—In the majority of healthy persons the systolic blood pressure lies between 110 and 135 mm, and the diastolic between 70 and 85 mm, the mean figure is about 120/80 Some healthy individuals have rather lower pressures, systolic readings of 100 to 110 are not uncommon As age increases there is a tendency for the pressure to rise from the lower to the higher of these limits, though many individuals retain a constant pressure despite advancing years

first sound. Pericardial friction is frequently affected by the amount of pressure used in applying the stethoscope; it can be made louder or fainter by varying the pressure. It is often transient, being present for a few hours and disappearing as an effusion develops. Sometimes it appears and disappears without apparent rhyme or reason.

Pleuro-pericardial friction is the term used when pericardial friction is heard only with those heart-beats which occur during a particular phase of the respiratory cycle Thus it may be audible only when the chest is in the position of expiration, only when it is in the inspiratory position, or only when it is passing through the mid position. Pleuro-pericardial friction can be a sign either of pericarditis or of pleurisy; it also occurs in mediastinitis.

Estimation of Blood Pressure and Examination of Arteries

The blood pressure should be taken as part of the routine examination of every case

Method.—The patient must be comfortably seated or lying with his arm resting on a low table. In some persons the blood pressure varies according to posture, some have a lower pressure when seated, others when lying: the difference may amount to 30 mm.: the position in which the pressure has been taken should therefore be recorded The patient must be relaxed, both physically and mentally. Some patients contract their biceps, clench their hands, tighten their abdominal, shoulder, or neck muscles immediately the cuff is inflated, any of these procedures vitiates the reading The arm and shoulder should be bare, the sleeve should never be merely pushed up, as this causes a tight band above the sphygmomanometer cuff and alters the reading. The room must be comfortably warm. cold affects the blood pressure, and it is useless to attempt a reading on a shivering patient. It is sometimes very difficult to get a patient to relax mentally, excitement, apprehension, and anxiety profoundly affect the reading. Frequently the true blood pressure can only be obtained after several readings when the patient has become accustomed to the procedure. Some patients must be examined on several different occasions. Occasionally a reflex rise of

on which initiates a reflex fall in blood pressure with loss of consciousness; the sensitivity of the sinus may result from general disease such as arteriosclerosis, or from tumours in the neck (carotid body tumours, glandular masses, etc.); in a predisposed individual the wearing of tight collars or sudden movements of the head often initiate the attack. Milder cases can be relieved by sedative such as phenobarbitone; more severe cases are sometimes cured by surgical denervation of the sinus, if a tumour is present, its removal may abolish the attacks. *Orthostatic hypotension* is characterised by a drop in blood pressure with faintness and weakness on assuming the erect posture. It can result either from "pooling" of blood in the veins, or from interference with the normal sympathetic vaso-constrictor reflexes. In the former case the heart rate is increased when the pressure drops, this type occurs in convalescence from acute illness, in anaemias, in pregnancy, and in persons with varicose veins or venous angiomata, it responds to treatment of the cause plus graduated exercise. The second variety occurs in disease of the central nervous system, especially with lesions of the hypothalamus, it is also seen after extensive sympathectomy. In this group the heart rate remains unchanged when the blood pressure falls. When the condition follows sympathectomy it tends to improve gradually in from 3 to 6 months, when secondary to cerebral disease it is extremely intractable but is occasionally helped by sleeping with the foot of the bed raised.

The Arteries.—The state of the arteries should be recorded in every case. In addition to feeling the radial arteries while counting the pulse, the brachial vessels should be palpated, at times the latter vessels are obviously thickened though the radial vessels are barely palpable. In health the arterial wall is not palpable, in arterial disease it is thickened, easily palpable, and harder, it is often nodular or tortuous. The retinal vessels should also be examined with an ophthalmoscope, in some cases there is gross disease of the peripheral arteries while the retinal arterioles remain normal, and vice versa. The earliest signs of retinal arteriolosclerosis is irregularity in calibre, at a slightly later stage segments of the arteries reflect light more brightly, where an arteriole crosses a venule, the latter is constricted. In more advanced disease the vessels appear like silver or copper wires with a bright line of reflection

Variations in Blood Pressure under Physiological Conditions.—Both the systolic and the diastolic pressures may be raised by muscular effort; in my own case I have recorded a rise from 112/78 at rest to 155/115 during strenuous exercise on a stationary bicycle. Failure to relax muscles, especially the abdominal muscles, will raise the pressure. The blood pressure is *also raised by excitement or anxiety*; these affect the systolic pressure more often than the diastolic, but sometimes the diastolic pressure is raised as well. Cold also raises the pressure. Finally, it is probable that there are daily variations in pressure, and that it is least during sleep.

Variations in Blood Pressure in Disease.—A raised systolic pressure with normal or low diastolic occurs in nervous excitement, thyrotoxicosis, aortic regurgitation, patent ductus arteriosus, arterio-venous aneurysm, and in sclerosis of the media of the arteries. A raised diastolic pressure is characteristic of hypertension provided muscular tension can be excluded; if the heart is efficient, the systolic pressure is raised in the proportion of 2 mm. for each 1 mm. rise in diastolic. A diastolic pressure which is persistently above 90 should be regarded with suspicion, and one which is persistently 100 or more is pathological. If the heart is inefficient, the rise in systolic pressure is proportionately less; if the arteries are rigid, it is proportionately greater.

Low blood pressure may be *persistent* or *transient*. A systolic level below 100 is almost always pathological, levels between 100 and 110 are suspicious. *Persistent hypotension* occurs in Addison's and Simmonds' diseases, severe myxoedema, and sometimes in acromegaly, it often follows coronary occlusion and is not infrequent in aortic stenosis but does not result from other forms of heart disease except in the terminal stages of failure. A low blood pressure in the absence of coronary occlusion, aortic stenosis, or advanced heart failure should never be attributed to a "weak heart". Treatment is that of the cause. *Transient hypotension* occurs in shock (p. 15), peripheral circulatory failure (p. 15), during paroxysms of cardiac arrhythmia especially the Stokes-Adams attacks of heart block (pp. 25 and 145), in epilepsy, and in hypoglycaemia. Simple faints and vaso-vagal attacks are associated with a transient reflex fall in blood pressure. *Carotid sinus syncope* is due to a hypersensitive carotid sinus, pressure

CHAPTER 4

X-RAY EXAMINATION OF THE HEART

In modern cardiology X-ray examination of the heart is becoming steadily more essential. It is true that there are many individuals in whom an accurate diagnosis can be made without an X-ray, and that there are many in whom the size of the heart can be gauged by palpation and percussion alone; but even in these cases X-ray gives additional information about the shape of the heart shadow which may be of considerable value in diagnosis and which cannot be obtained by any other means. There are a great many cases in which the diagnosis remains uncertain in the absence of X-ray examination

In cardiac cases, in addition to screening the patient, films should be taken with a minimum distance of 6 feet between the tube and the film, if the distance is shorter than this, the heart shadow is enlarged; and the nearer the tube to the film, the greater is the degree of distortion. In lung radiology films are frequently taken at a distance of 3 to 4½ feet in order to secure better detail, a misleading idea of the size of the heart may be obtained from a film which has been taken at this distance A film taken with a portable unit in a bedridden patient is almost valueless as an index of heart size, not only is the distance short, but the shape of the heart shadow in a recumbent or semi-recumbent patient is not in the least comparable with its shape in the erect position

In routine cardiac radiology films are exposed with the patient erect and a 6-foot film-tube distance. In this country it is customary to make the exposure at the end of a full inspiration, in some other countries the exposure is made with the chest midway between inspiration and expiration. When comparing films which have been made from a given patient on different dates, it is important first to make sure that the films are comparable, the two films should be placed one over the other so that the clavicles are superimposed, the films are not comparable unless the shadows of the ribs and of the two leaves of the diaphragm coincide on each film.

running along their centre. At times the artery may narrow to a mere thread along part of its course. In still more advanced cases haemorrhages are seen in the retina, and glistening white spots known as "fish-scale patches"; the haemorrhages tend to be linear or flame-shaped ("hypertensive retinitis").

Ballistocardiography

A ballistocardiogram is a graphic record of the movement imparted to the tissues by the systolic and diastolic rushes of blood through the great vessels. A simple apparatus consists of two coils of wire connected in series and mounted in a magnetic field; these are strapped to the patient's shin. The magnet is placed between the coils and kept stationary by fixing it to the examination table. Tissue movements are imparted to the coils; and as these move in relation to the stationary magnet, currents are set up and recorded by a standard cardiograph.

It is claimed that a ballistocardiogram will distinguish high output failure from failure with normal or low output; and that it will help in the recognition of coarctation of the aorta, coronary insufficiency, and shock, as well as in detecting those cases of angina which are likely to benefit from an elastic abdominal belt.

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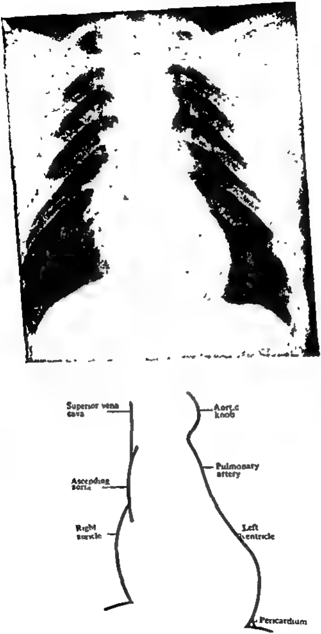

FIG. 1.—Teleradiogram showing normal heart shadow, antero-posterior view. Average axis. For description, see text. From a man aged 38.

It is extremely difficult to get really comparable films; the slightest difference in the depth of inspiration alters the height of the diaphragm. A raised diaphragm throws the apex of the heart outwards and upwards, making the heart appear larger. It should be remembered too that an exposure made during systole will show a smaller shadow than one made during diastole, unless special means are taken to record the cardiogram while the exposure is being made, and to mark the moment of exposure on the latter, there is no means of telling whether a given exposure has been made in systole or in diastole. The difference between the systolic and diastolic size of the heart shadow may amount to as much as a centimetre on either side, making two centimetres in the transverse diameter It will be realised, therefore, that great care must be taken before conclusions are drawn from an apparent change in the size of the heart shadow on different dates; slight changes which require measurement for their demonstration are almost certainly devoid of significance; provided the films are comparable, changes in shape or size which can be seen at a glance are significant.

The Antero-posterior View (Figs 1-3).—The following bony structures should first be identified. The vertebrae lie in the middle line until they become hidden by the mediastinal shadow. The posterior aspects of the ribs run downwards and outwards, their anterior aspects curve downwards and inwards The clavicles run outwards and a little upwards across either side of the upper part of the chest, forming a slightly sinuous curve. If the film is truly antero-posterior, the spines of the vertebrae lie exactly midway between the inner ends of the clavicles, should this not be the case, the view is semi-right or semi-left oblique, and a corresponding change in the shape of the heart shadow is produced: with rotation towards the patient's left (towards the right oblique position) the aortic knob gradually disappears while the pulmonary conus becomes at first more prominent, with rotation in the reverse direction the aortic knob becomes more prominent and the right cardiac contour becomes hidden. Scoliosis also alters the shape and position of the heart shadow, and should be looked for before the latter is assessed. Where the vertebrae are hidden by the mediastinal shadow, scoliosis can be detected by an alteration in the direction of the ribs on the two sides

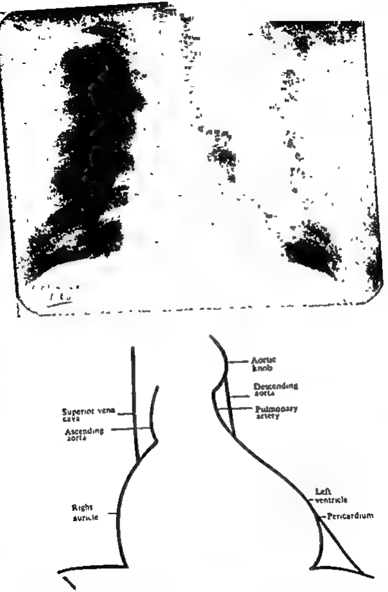

Fig 3.—Normal heart, transverse type, antero posterior view From a well-developed, heavily built, rather stout man aged 43 with mild bronchitis but no evidence of organic heart disease. Note the short broad chest, flat diaphragm, and rounded apex of left ventricle seen within the pericardial shadow TD heart, 15 4 cm ; chest 34 5 cm

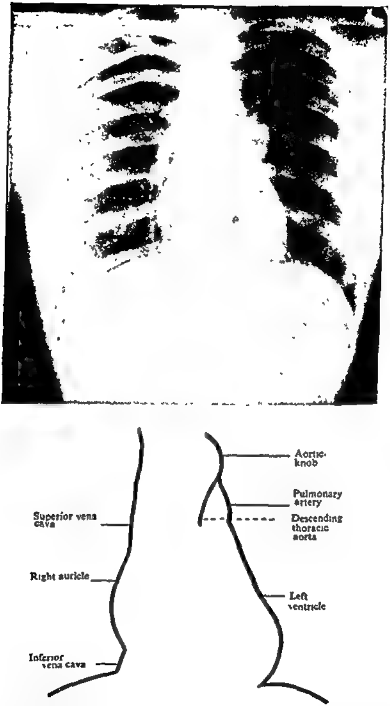

FIG. 2.—Teleradiogram showing normal heart shadow, vertical type In this film the right border of the ascending aorta does not project beyond the superior vena cava, which forms the right border of the mediastinal shadow down to its point of junction with the right auricle Female, aged 22.

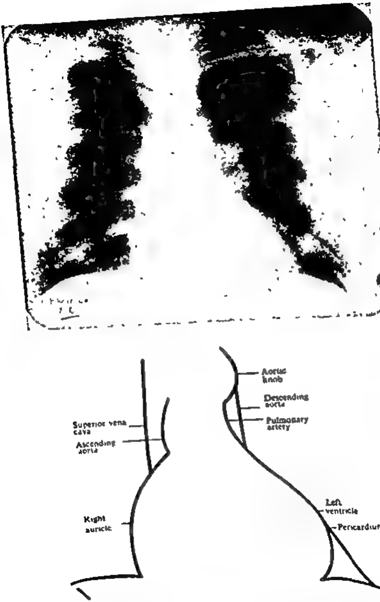

FIG 3—Normal heart, transverse type, antero-posterior view. From a well developed, heavily built, rather stout man aged 43 with mild bronchitis but no evidence of organic heart disease Note the short broad chest, flat diaphragm, and rounded apex of left ventricle seen within the pericardial shadow TD heart, 15 4 cm, chest 34 5 cm

and by widening of the interval between them on one side. The height of the diaphragm on each side should also be noted.

The right border of the mediastinal shadow is formed by the superior vena cava, right auricle, and right ventricle from above downwards; in some films the ascending aorta also takes part, projecting beyond a portion of the vena cava. The *superior vena cava* is seen as a straight border running downwards from the right sterno-clavicular joint; it is sometimes obscured by the sternum. The *ascending aorta* in its upward course has a gentle convexity to the right; its right border is often seen through the shadow of the vena cava, slightly medial to it; but in some films it projects beyond the vena cava and obscures a portion of it. The *right auricle* produces a prominent convexity which joins the superior vena cava at an angle; the lower third of this convexity is formed by the *right ventricle*, but the point of junction of auricle and ventricle cannot be determined on a plain film, a kymogram being needed for this purpose. The lower end of the right cardiac border meets the shadow of the right leaf of the diaphragm at an acute angle; the *inferior vena cava* can sometimes be seen cutting across the apex of this angle. About midway down the right border, the branches of the pulmonary artery and vein emerge from behind the superior vena cava and aorta to enter the right hilar shadow.

The left border of the mediastinal shadow is formed by three arcs. The uppermost is the aortic knob produced by the *arch of the aorta*; from it the fainter shadow of the descending aorta can be traced downwards until it becomes hidden by the remainder of the cardiac shadow. The second arc, known as the "left middle arc" or pulmonary arc, is formed by the *conus pulmonalis* and *pulmonary artery*; the branches of the left pulmonary artery run outwards from behind it into the left hilum; the branches of the right pulmonary artery are hidden by the aorta until they emerge from behind its right border to enter the right hilum. The third or left lower arc is the largest and most prominent; it is formed by the *left ventricle*. At its junction with the middle arc a small portion is formed by the left auricular appendix; unless the latter is enlarged, this portion cannot be distinguished from the remainder. It should be noted that, except for this small

portion of the left lower arc, the left auricle takes no part in producing the cardiac outline in the antero-posterior view; it is lying posteriorly, hidden by left ventricle and conus. The apex of the heart is rounded and forms an acute angle with the diaphragm; but in some films the pericardium runs downwards and outwards from the apex to form an obtuse angle with the diaphragm; its shadow is less dense than that of the ventricle, the outline of which can be seen within it (Fig 3)

The general shape of the chest makes a considerable difference to the appearance of the heart shadow. An acquaintance with the normal variations is an essential preliminary to any consideration of pathological appearances. In persons with a long narrow chest (asthenic type) the whole mediastinal shadow is relatively more vertical and narrower ("vertical heart"), the aortic knob tends to be small and the descending aorta may be more central and hidden, the pulmonary conus tends to be relatively prominent (Fig. 2). In thick-set individuals with a short broad chest (sthenic type) and in the obese the long axis of the heart shadow runs more transversely, occasionally almost horizontally ("transverse heart"), while the height of the whole mediastinal shadow is shortened. The ascending aorta tends to be more convex to the right and the aortic knob to be more prominent on the left, the left middle arc is less prominent, the left ventricular contour extends further to the left (Fig 3) It is important not to mistake a transverse heart shadow for one showing enlargement of the left ventricle, or a vertical heart for one showing enlargement of the pulmonary conus As will be seen later (Chapter 6), corresponding variations occur in the electric axis of normal cardiograms

In the right oblique view (Fig 4) the spine appears to the left, the mediastinal shadow lying anterior to it. The anterior border of the heart shadow is formed in its lower part by the right ventricle and above this by the pulmonary conus. Above the pulmonary conus the aorta runs a short distance upwards before curving backwards towards the spine, the aortic shadow is often poorly defined in this view The innominate artery and superior vena cava, if visible, continue the original line of the aorta upwards The pulmonary artery lies beneath the concavity of the aortic arch and behind that portion of the cardiac

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shadow formed by the conus; it is usually relatively denser and better defined than the shadow of the aortic arch. As a rule the posterior border of the mediastinal shadow is ill defined, but it can be outlined by making the exposure while the patient is swallowing thick barium paste so as to fill the oesophagus with barium. Normally three shallow impressions are seen on the anterior border of the oesophagus. The uppermost or aortic impression is produced by the arch of the aorta. The middle impression is produced by the pulmonary artery. The lower impression is produced by the left auricle, which forms the posterior border of the cardiac shadow in this view.

In the **left oblique view** (Fig. 5) the spine appears to the right. The anterior border of the heart shadow is formed in its lower part by the left ventricle, above this by the pulmonary conus as it crosses the root of the aorta, and above the conus by the ascending aorta. From the aortic shadow, the innominate, left carotid, and left subclavian arteries continue the shadow upwards while the arch of the aorta curves backwards towards the spine and continues downwards overlying the shadows of the vertebrae as the descending thoracic aorta. The clear space enclosed beneath the aortic arch is known as the *aortic window*, the clear space above the aortic arch, bounded by the upper border of the latter, by the anterior margins of the vertebral bodies, and by the left subclavian artery, is known as the *aortic triangle*. The posterior border of the heart shadow is clearly defined and rounded in its lower two-thirds, which are normally formed by the left ventricle. The upper third, connecting this to the lower border of the aortic arch, is rendered obscure by the left hilar shadow, from which the branches of the left pulmonary artery and vein radiate into the lung, some of these cross the aortic window, while others (seen in sectional view) appear as rounded shadows. The trachea runs downwards as a translucent band in the upper part of the chest, the left bronchus continues this band downwards, passing behind the aorta to enter the aortic window. When the left auricle is enlarged, it projects behind the left ventricle and displaces the oesophagus backwards.

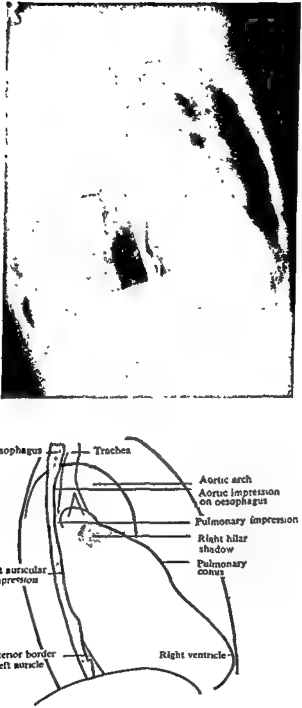


FIG. 4.—Teleradiogram showing normal heart shadow, right oblique view Oesophagus filled with barium. Male, aged 53

Right auricular enlargement is seen in the antero-posterior view, where it produces increased convexity of the right cardiac contour (Fig. 8). It is the lower of the two arcs which is affected. This contour is also rendered more convex by a transverse lie of the heart (Fig. 3).

Right ventricular enlargement gives rise in the first instance to enlargement of the conus pulmonalis, visible in all three views. In the antero-posterior view the left middle arc becomes more prominent; with minor degrees of enlargement it fills up the space between the aortic knob and the left ventricular contour, so that the left cardiac border runs in a more or less straight line from the aortic knob to the apex (Fig. 6), with greater enlargement the left middle arc projects, causing the whole heart shadow to appear more square (Fig. 8 (A)) In the right oblique view the anterior heart border is more rounded, and there is often a definite forward bulge towards its upper part where the conus is passing into pulmonary artery (Fig 9) In the left oblique view the enlarged conus and pulmonary artery also produce a bulge in this area. These appearances are seen in mitral stenosis, in pulmonary stenosis, in atrial septal defect, and in chronic lung diseases, especially emphysema. Mitral stenosis is distinguished from the remaining conditions which cause enlargement of the conus by the presence of left auricular enlargement as well (Figs 7 and 9)

Left auricular enlargement is best demonstrated in the right oblique view where it is shown by a marked increase in the auricular impression on the oesophagus The oesophagus shows a sudden change in direction backwards just below the

auricular enlargement the starting-point of the backward displacement is higher up Enlargement of the left auricle produces no change in the antero-posterior view until it reaches a considerable degree, when distension of the left auricular appendix gives rise to a projection on the left border between the pulmonary and left ventricular contours (Fig 8); this is sometimes seen in mitral stenosis in place of, or in addition to, enlargement of the pulmonary artery and conus With gross enlargement of the left auricle, it expands to the right behind the right auricle and in these circumstances it may project

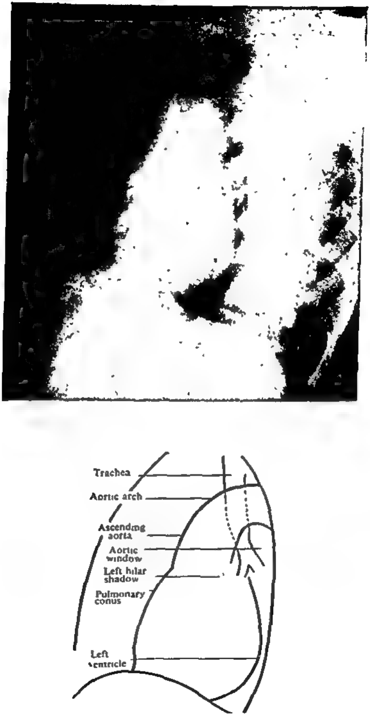


FIG. 5.—Teleradiogram in left oblique position. Normal heart shadow. Male, aged 38.

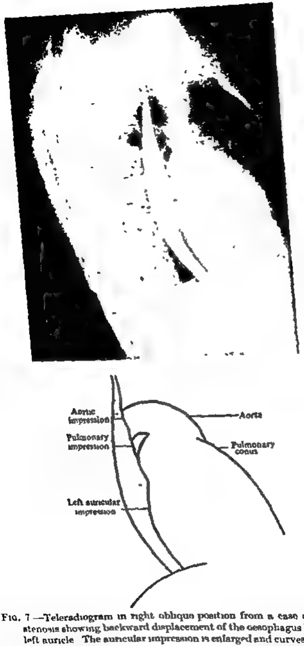


FIG. 7.—Teleradiogram in right oblique position from a case of early mitral stenosis showing backward displacement of the oesophagus by an enlarged left auricle. The auricular impression is enlarged and curves back abruptly just below the pulmonary impression. From a man of 30 with history of recurring haemoptysis; there was a constant systolic murmur at the

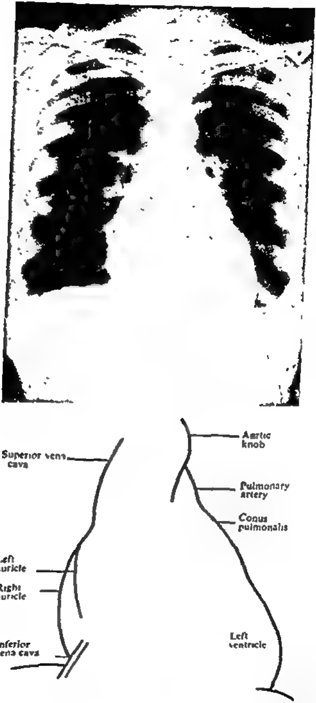
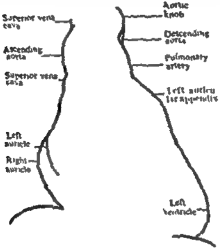


Fig 6.—Antero-posterior teleradiogram in mitral stenosis. Female, aged 20, complaining of palpitation due to paroxysmal tachycardia. The prominent conus pulmonalis is visible on the left border. The outline of the enlarged left auricle is faintly seen through the right auricular shadow. The inferior vena cava is seen crossing the cardio-hepatic angle. There is no pulmonary congestion.

beyond the latter so as to form part of the right heart border in the antero-posterior view. Enlargement of the left auricle is seen particularly in mitral stenosis, and is especially useful in distinguishing between organic and secondary or innocent murmurs in the mitral area. Left auricular enlargement may also occur later as a sequel to left ventricular enlargement.



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Left ventricular enlargement is seen in the antero-posterior and in the left oblique views. In the former the left lower arc becomes more prominent, more convex, and altogether more bulky-looking (Fig. 10). In assessing this, due allowance must be made for the type of chest. The "Heart/Chest ratio" may be of assistance for this purpose; normally, the transverse diameter of the heart shadow is not greater than one-half of the transverse diameter of the chest at the level of the diaphragm. The transverse diameter of the heart is measured by taking the most prominent point on the right and left borders, measuring the distance of each from the middle line, and adding the two distances together. With left ventricular enlargement the ratio usually becomes greater than 1–2. When the hypertrophy of the left ventricle is *concentric*, as in hypertension or aortic stenosis, the displacement of the apex is directly outwards so that the whole mediastinal shadow becomes *more*

FIG. 8 – Antero-posterior teleradiogram in mitral stenosis. Female aged 44 with auricular flutter. The grade of mitral stenosis is more advanced than that shown in Fig. 6. The aortic knob is small; immediately beneath it the left middle arc is slightly convex instead of concave, indicating enlargement of the pulmonary conus, while the main branches of the pulmonary artery radiating from the hila are distended; there is a marked projection on the left border occupying a position between the pulmonary and left ventricular arcs, caused by the distended left auricular appendix; the right auricular contour is displaced to the right and is more convex than normal in consequence of enlargement of the right auricle, and the outline of the left auricle can be seen faintly through it. The left ventricle is also slightly enlarged as a result of simultaneous mitral regurgitation. The opacity in the right costo-phrenic angle is the remains of a resolving pneumonic consolidation. The right oblique view from this case is shown in Fig. 9.

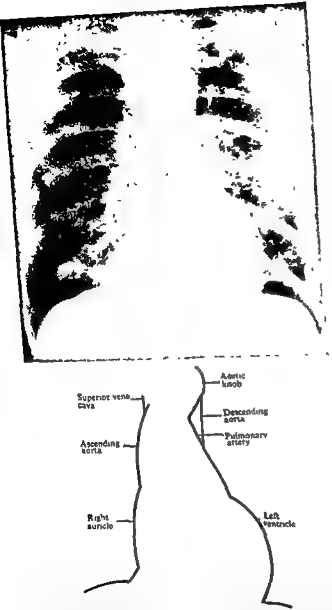


FIG 10.—Concentric hypertrophy of the left ventricle in hypertension. From a man of 55 with diabetes, blood pressure 235/150, and forcible heaving impulse. Although the heart/chest ratio is well within the limit of 1-2, the left ventricular contour shows a marked increase in convexity. The lung fields are congested

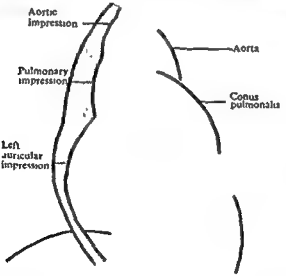


FIG. 9.—Right oblique teleradiogram in advanced mitral stenosis. From the same case as Fig. 8. The prominent conus pulmonalis is seen anteriorly, while the oesophagus is displaced backwards by the enlarged left auricle.

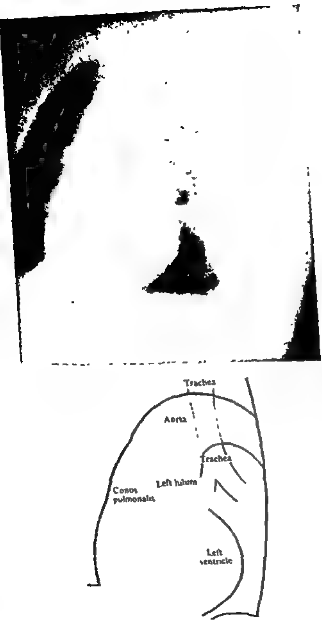


FIG 12 —Left oblique teleradiogram showing enlargement of left ventricle. Male, aged 46, with essential hypertension, BP 214 136, sclerosis of retinal arteries and enlargement of left ventricle Note the wide aorta and prominent left ventricular contour

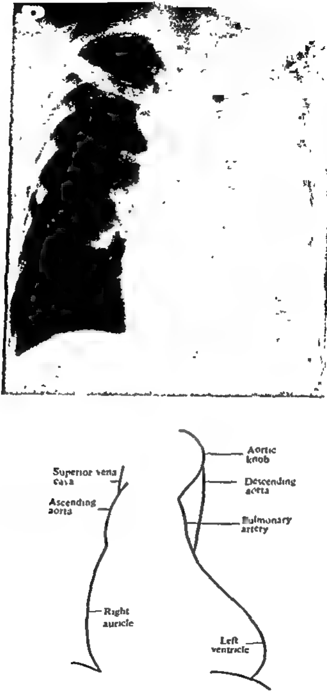
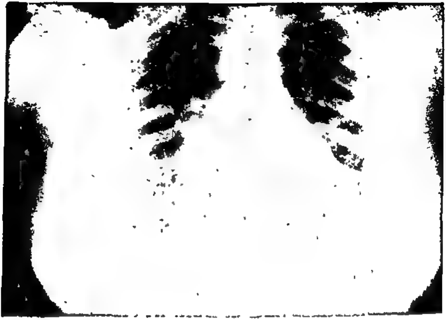


Fig. 11.—Hypertrophy and dilatation of left ventricle in aortic regurgitation

angular or "boot-shaped" (Fig. 10). In aortic regurgitation the ventricle is dilated as well as hypertrophied; the displacement of the apex is downwards and outwards, so that the heart shadow appears elongated as well as enlarged to the left (Fig. 11). In the left oblique view the posterior border of the heart shadow becomes more prominent and bulky-looking (Fig. 12)

Combined Lesions and Generalised Enlargement.—The combination of mitral stenosis with aortic regurgitation is frequent, and it produces the radiological signs of both lesions, viz enlargement of the left ventricle and enlargement of the conus (Fig 13). Various conditions lead to generalised enlargement, the whole cardiac shadow becoming enlarged while its general shape remains normal; these conditions include coronary disease, the later stages of thyrotoxicosis, myxoedema, and auricular fibrillation (Fig 14). Pericardial effusion leads to generalised enlargement but the shape of the heart is altered, becoming more pear-shaped or globular (Fig 15), occasionally a double contour can be seen in cases of pericardial effusion, on screening, the amplitude of pulsation is small

Other Radiological Findings in Cardiovascular Disease.—Myocardial infarction which has led to an *aneurysm of the heart* gives rise to a localised bulge in the affected region, most often the left ventricle, this may show up in one of the standard views, or it may be necessary to expose a film at an appropriate angle which must be determined by preliminary screening. *Aneurysm of the aorta* appears as a localised bulge or general widening of the aortic shadow at some part of its course, it must not be confused with *tortuosity of the aorta* which occurs in arteriosclerosis and in hypertension, and which leads to increased convexity of the right border, where it is formed by the ascending aorta coupled with increased prominence of the aortic knob *Calcified plaques* are often seen in the aorta in cases of arteriosclerosis *Calcification of the aortic valve cusps* can sometimes be recognised. The radiological appearances of venous congestion of the lungs have been described on p. 28 and are shown in Fig. 10, the remaining conditions mentioned are illustrated in later chapters



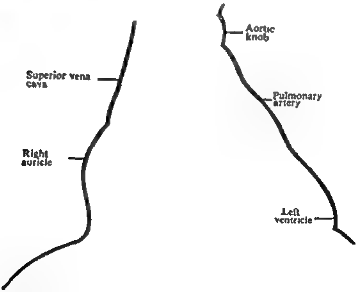


Fig. 13.—Combined mitral and aortic valvular disease. The left ventricle, pulmonary conus, and right auricle are all enlarged. The lung fields are congested. From a female, aged 26

Angiocardiography

Angiocardiography is a method of outlining the individual cardiac chambers, the pulmonary artery and its branches, and, in favourable circumstances, the aorta. A radio-opaque dye (70 per cent Diodone, or 70 per cent Diodrast) is injected intravenously,

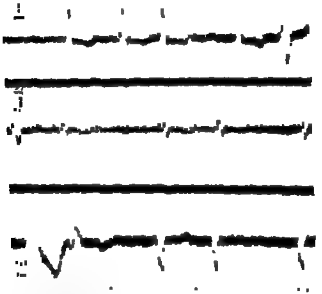


Fig. 14.—Generalised cardiac enlargement. From a man of 51 with thyro-toxicosis and auricular fibrillation complicating hypertension. Th[illegible]

40 c.c. being required in the case of an adult. The [illegible]

[illegible] X-ray exposures are made at predetermined intervals after the start of the injection with a view to outlining the right auricle and ventricle (1 to [illegible] seconds), the right ventricle and pulmonary artery (2 to 3 seconds), and the left ventricle with aorta (4 to 8 seconds) on successive films. A

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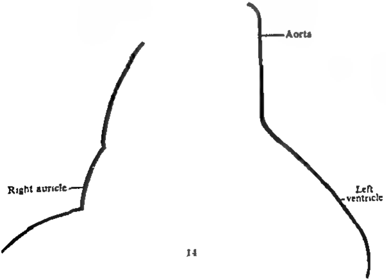

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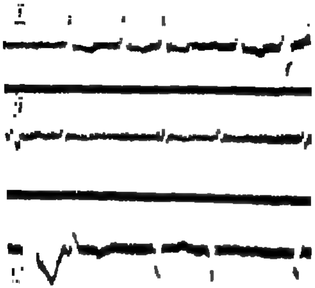

Fig 14 —Generalised cardiac enlargement From a man of 51 with thyrotoxicosis and auricular fibrillation complicating [illegible]

40 c.c. being required in the case of an adult. The [illegible] is commonly made into the median basilic vein and the total [illegible] must be given quickly Better results have been obtained [illegible] by using the external jugular vein. X-ray exposures are [illegible] predetermined intervals after the start of the injection [illegible] to outlining the right auricle and ventricle (1 to 2 [illegible] right ventricle and pulmonary artery (2 to 3 seconds [illegible] left ventricle with aorta (4 to 8 seconds) on [illegible]

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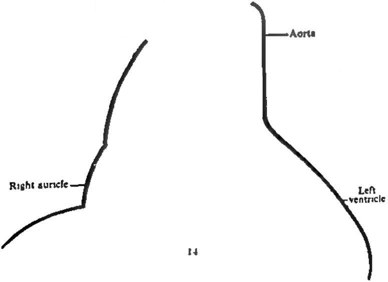

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Taussig reports a 1 per cent mortality even when all precautions have been taken. Various reactions to injection of the dye may occur, particularly in sufferers from asthma and other allergic diseases, patients with a history of any such condition should not be subjected to angiocardiography, and in all patients it is advisable to carry out a preliminary test for sensitivity either by instillation of dye into the conjunctiva or by preliminary injection of 0.5 c.c. intravenously. Apart from anaphylactic reactions, death has occurred in a small number of patients with cyanotic congenital heart disease.

Kymography

A metallic radio-opaque grid with a series of transverse slits at intervals of 1 cm. is interposed between the X-ray tube and the film. A long exposure is made (1 to 2 seconds, depending on the patient's heart rate), and during the exposure the grid is made to move uniformly downwards for a distance of 1 cm. The resulting film shows

to the exp

the period

occurred,

each strip has a wavy outline; the waves represent the systolic and diastolic movements of the corresponding portion of the heart.

A kymogram allows the various structures which take part in forming the cardiovascular shadow to be identified with certainty, since auricles, ventricles, arteries, and veins each give a different wave form with different time relationships. It also enables the amplitude of the cardiac pulsations to be measured; it may show absence of pulsation over an area which is the seat of infarction, or generalised reduction in amplitude of pulsation due to pericardial thickening or effusion. Shirley Smith claims to have demonstrated additional and characteristic waves in cases of patent ductus arteriosus, and he considers that kymography will eliminate errors in the diagnosis of patent ductus.

Tomography

During an X-ray exposure the tube is made to move a short distance in one direction while the film moves in the

direction

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it are th

varying the relative movements of

mechanical contrivance for changing the X-ray cassettes allows a greater number of exposures to be made in the ten seconds following the injection; but good results can be obtained with hand-changing of the cassettes by an expert team If oblique or

FIG 15.—Enlargement of the cardiac shadow, due to pericardial effusion [illegible] and mitral valvular lesions [illegible] s of the individual chambers

lateral views are required in addition to postero-anterior views, two X-ray tubes may be used simultaneously so as to avoid the necessity of a second injection

The method is of particular help in the study of congenital heart disease It is also useful in other cases in which the interpretation of plain X-ray films is difficult. It should not be used indiscriminately, as the procedure is not devoid of danger; Dr. Helen

CHAPTER 5

CARDIAC RHYTHM

The normal cardiac rhythm originates in a node of specialised tissue known as the *sino-auricular node*. It lies in the wall of the right auricle close to the point of entry of the superior vena cava. It has the property of automatic stimulus production; when severed from all nervous connections (as in the excised perfused heart) it will continue to excite rhythmic contractions of the auricles at a rate of about 70 per minute. In the intact organism its rate of stimulus production is modified by the central nervous system; it is increased by activity of the sympathetic (accelerator), diminished by activity of the vagus (inhibitory). The stimuli produced by the sino-auricular node excite *contraction of the auricles*.

The *auriculo-ventricular node* is a second collection of similar tissue lying in the interauricular septum just above the auriculo-ventricular septum. It too has the capacity to produce stimuli automatically; if isolated from the sino-auricular node ("first Stannius ligature" in animals, "Nodal Escape" and "Nodal Rhythm" in human beings) it produces stimuli at a rate of 50 to 60 per minute and controls the *rhythm of the ventricles*. In ordinary circumstances, however, the auriculo-ventricular node is excited by contraction of the auricles in response to the sino-auricular node. The automatic rate of the S–A node being greater than that of the A–V node, the automatic action of the latter has no opportunity to function. With normal rhythm, therefore, the S–A node controls the rhythm of the whole heart. The automatic action of the A–V node provides a safety mechanism which may come into play if the S–A node fails or becomes unduly slowed ("Nodal Escape", see Fig. 21).

From the auriculo-ventricular node the *auriculo-ventricular bundle* (A–V bundle or Bundle of His) *arises*. *It forms the only muscular connection between the auricles and the ventricles* which are everywhere else separated by the fibrous auriculo-ventricular septum. Some authorities maintain that a second connecting bundle (or series of strands) known as the "Bundle

the tube and film it is possible to photograph a series of successive planes lying at differing depths from the anterior body wall.

Tomography has found its chief application in diseases or injuries of the lungs. It can be used to locate the depth of a foreign body from the surface, to show up cavities which are masked in ordinary films by structures lying in front of them or behind them, to outline the bronchi and demonstrate bronchial stenosis, or to demonstrate tumours, etc. It has been less extensively used in cardiology hitherto; but patent ductus arteriosus has recently been successfully outlined by this method, and it seems likely that it may prove useful in the study of other anomalies, particularly those involving pulmonary vessels. It may also prove of value for the localisation of foreign bodies in the heart or pericardium.

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CHAPTER 5

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of Kent" exists in certain individuals (see p. 120); its existence is not universally accepted. Having passed through the A–V septum, the bundle divides into *right and left branches* which lie astride the interventricular septum. The left branch subdivides almost immediately; its ramifications spread over the left ventricle and are ultimately distributed to the muscle through the Purkinje network of fibres. The right branch of the bundle runs downwards as a single strand for about an inch towards the papillary muscle; then it too subdivides and is distributed over the right ventricle. *With normal rhythm* the function of the A–V bundle is conduction. The rate of conduction, though less rapid than in nerve fibres, is considerably more rapid than through muscle. The stimulus from the A–V node is rapidly distributed all over each ventricle, thus ensuring that the ventricular contraction follows shortly after the auricular contraction, that the two ventricles contract simultaneously, and that the contraction of each is concentric. The time which elapses between the liberation of the S–A stimulus (commencement of auricular systole) and the commencement of ventricular systole averages 0·16 second (ranging from 0·12 to 0·20 second); it is known as the conduction time (PR interval in cardiograms, a–c interval in polygrams), it includes the time taken for the wave of excitation to pass over the auricles (0·06 to 0·08 second) and for it to be conducted down the A–V bundle (0·08 to 0·10 second). The interval which elapses between the onset of contraction in the first and last parts of the ventricles to be stimulated lies between 0·06 and 0·08 second.

The A–V bundle, like the A–V node, has the power of automatic stimulus production. The automatic rate becomes steadily lower as the bundle is descended until, in its lower reaches, the rate is from 30 to 35 per minute. In ordinary circumstances this automatic action is never given a chance to function; but if the passage of stimuli from the A–V node along the bundle is interrupted, a point in the latter below the block will initiate the rhythm of the ventricles ("Second Stannius ligature" in animals, "Heart block" in human subjects).

Both the A–V node and the A–V bundle are subject to control by the vagus and sympathetic; but the extent of the control steadily lessens from the node downwards, and the

nervous control of the A–V node is less than that of the S–A node

Cardiograms

When muscle contracts, it produces electrical changes; a cardiogram is a record of the electrical changes which accompany cardiac contraction. They can be recorded directly by using a sensitive galvanometer (Einthoven's string galvanometer), the fibre is suspended in a beam of light, and movements of its shadow are recorded after magnification on a moving photographic film or plate Alternatively the currents generated by cardiac activity are amplified before being recorded by a galvanometer or a cathode ray oscillograph. Whichever instrument is used, it is standardised, so that an E M F of 1 millivolt produces a deflection of 1 cm' in the finished record and a time marker is introduced which, with most cardiographs, measures fifths and twenty-fifths of a second, the finer lines are 0 04 second apart, the thicker ones 0 2 second

Leads are taken from the right arm, left arm, and left leg. The record obtained using the right arm–left arm leads is lead 1 That using the right arm–left leg leads is lead 2; while the left arm–left leg leads give lead 3 The connections are so made that a current in the direction right arm–left arm, right arm–left leg, or left arm–left leg gives an upright deflection. For the study of cardiac rhythm these standard limb leads usually suffice, but for the investigation of myocardial disease a series of chest leads is often required as well, since the limb records alone may prove misleading The method of recording chest leads, and their interpretation is described in Chapter 6

Normal Cardiograms.—The normal record shows a rounded or pointed upright deflection known as "P" produced by auricular systole After a short interval (the PR interval) this is followed by a series of narrow pointed deflections known as "QRS" resulting from the onset of ventricular systole; Q is usually small and directed downwards, R large and upright, S smaller and downwards QRS is followed by another interval during which the galvanometer fibre is iso-electric and the record is stationary on the base line. Finally, there is a rounded upright deflection "T" and associated with the termination of ventricular systole (Fig 16, A) After an

of Kent" exists in certain individuals (see p. 120); its existence is not universally accepted. Having passed through the A–V septum, the bundle divides into right and left branches which lie astride the interventricular septum. The left branch subdivides almost immediately; its ramifications spread over the left ventricle and are ultimately distributed to the muscle through the Purkinje network of fibres. The right branch of the bundle runs downwards as a single strand for about an inch towards the papillary muscle; then it too subdivides and is distributed over the right ventricle. With normal rhythm the function of the A–V bundle is conduction. The rate of conduction, though less rapid than in nerve fibres, is considerably more rapid than through muscle. The stimulus from the A–V node is rapidly distributed all over each ventricle, thus ensuring that the ventricular contraction follows shortly after the auricular contraction, that the two ventricles contract simultaneously, and that the contraction of each is concentric. The time which elapses between the liberation of the S–A stimulus (commencement of auricular systole) and the commencement of ventricular systole averages 0·16 second (ranging from 0·12 to 0·20 second); it is known as the conduction time (PR interval in cardiograms, a–c interval in polygrams), it includes the time taken for the wave of excitation to pass over the auricles (0·06 to 0·08 second) and for it to be conducted down the A–V bundle (0·08 to 0·10 second). The interval which elapses between the onset of contraction in the first and last parts of the ventricles to be stimulated lies between 0·06 and 0·08 second.

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Both the A–V node and the A–V bundle are subject to control by the vagus and sympathetic; but the extent of the control steadily lessens from the node downwards, and the

in lead 3, but gross notching is pathological. The interval between QRS and T is iso-electric, that is to say, level with

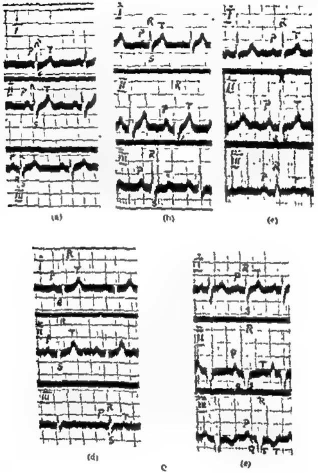


FIG. 16.—Th[illegible] ...ads 2

the base line, sometimes it is very short, especially with rapidly beating hearts. T is upright in leads 1 and 2, but is sometimes inverted in lead 3 The beats are evenly spaced.

interval corresponding to diastole, the whole series is repeated. Some records show a small wave "U" following T in the early phase of diastole; its significance is unknown.

In a normal record (Fig. 16, A, B, C) there is a regular sequence of auricular and ventricular complexes; each P is

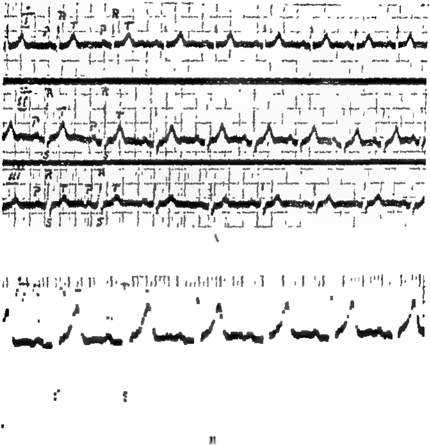


Fig. 16 The normal electrocardiogram

A *Normal cardiogram, standard limb leads* Full length

B *Normal cardiogram, chest lead 4R (apex- right arm)*

followed by its own QRS and T P is always upright in leads 1 and 2, and usually also in lead 3, but it is occasionally diphasic or inverted in the latter lead The PR interval, measured from the beginning of P to the beginning of QRS, does not exceed 0·2 second The breadth of QRS does not exceed 0·10 second. Q or S or both may be absent in one or all leads. Slight notching of QRS may be present, especially

tension, in myxoedema, and in cerebral compression It is diminished or abolished when the heart rate is increased; so it disappears after exercise, during fevers, etc. It is a normal physiological response to the effect of inspiration on the venous return It is never a sign of heart disease; it is equally not a sign that the heart is free from disease, though it does show that the reflex mechanism, whereby distension of the right auricle causes tachycardia, is intact, and that the *cardiac rhythm* is normal

Other varieties of sinus arrhythmia. At times there are variations in heart rate which occur apart from the respiratory movements or from exercise They are not infrequent in emotional patients who may show an unaccountable and transient rise or fall in heart rate Sometimes the movement of swallowing causes a relatively sudden fall in heart rate followed by a gradual return to the original level. These changes in rate are not pathological, but it is necessary to distinguish them from pathological irregularities in rhythm.

Sino-auricular Block

Sino-auricular block is an extreme variety of sinus arrhythmia in which slowing due to vagus action produces an interval almost double that between the previous two beats, so that it seems as though a beat were missed altogether. Some authors consider it the result of disease of the auricular muscle whereby a sinus stimulus is prevented from reaching the auricle. With this view, however, I disagree The condition is by no means infrequent in patients whose hearts are healthy, and it appears to be due to sudden activity of the vagus This is sometimes reflex, due to irritation of the stomach or pleura, or to swallowing, it is often emotional, in occasional cases it may be toxic and ...

... to activity of the vagus, though in these cases the possibility of an organic block must be admitted.

Sino-auricular block usually causes no symptoms Occasionally it is associated with a transient fall in blood pressure and faintness or actual syncope, these being other features of vagal

Inspiration increases the venous return to the chest, and hence produces greater filling of the right auricle. Some persons respond to this by increasing the output per beat, others by increasing the heart rate. This increase in rate is reflex, brought about by distension of the right auricle (Bainbridge's accelerator reflex). Some individuals combine the two methods. Both are physiological. When the response

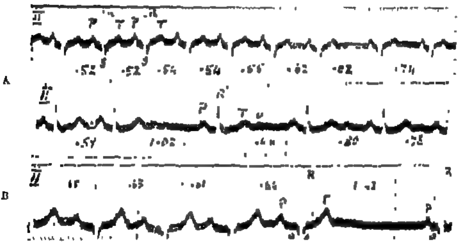


FIG. 17.—Sinus arrhythmia and sino-auricular block.

A. *Sinus arrhythmia.* Examples from two different patients. Lead 2. The interval between successive beats is noted in seconds. A small U wave is seen following T in the second example.

B. *Sino-auricular block.* The interval between successive beats is noted in seconds.

involves an increase in rate the resulting irregularity of the heart and pulse is called *respiratory sinus arrhythmia.* The heart accelerates shortly after the beginning of inspiration, and it slows soon after the start of expiration. There is alternately an increase and decrease in heart rate accompanying the respiratory movements, and the changes in rate are gradual. Cardiograms (Fig. 17, A) show corresponding variations in the spacing of the beats, the diastolic intervals being shorter during inspiration and longer during expiration. The sequence of the deflections is unchanged, and the individual deflections are unaltered. Respiratory sinus arrhythmia is especially frequent in children; it is also common in adults with slow heart rates: thus it occurs in athletes, in hyper-

A fall in the carotid pressure is the explanation of the tachycardia in *shock*, in *haemorrhage*, in conditions of *dehydration*, and in *left heart failure*

(2) **Central Stimulation of the Sympathetic Centres in the Medulla.**—*Bacterial toxins* are frequently responsible for central stimulation of the sympathetic with resulting tachycardia; thus tachycardia is present in most *acute infective illnesses* (with a few exceptions such as typhoid), and in many *chronic infections* such as tuberculosis, chronic pyuria, etc. Tachycardia often persists for some time during *convalescence* from acute fevers, especially influenza; it would seem that stimulation of the centres during the acute stage has left them more sensitive to accelerator and less sensitive to inhibitory reflexes, a process which presumably corresponds to Sherrington's "facilitation of reflexes"

Emotion is another very common cause of tachycardia, perhaps the most frequent cause of all. The emotions are closely linked with the medullary centres subserving the vegetative nervous system, and they are capable of increasing either vagal or sympathetic tone, more often the latter. *Anxiety states* are very frequently responsible for a persistent tachycardia, and not infrequently for an erroneous diagnosis in consequence, it should be remembered that the presence of an organic cardiovascular lesion does not render the victim immune from anxiety, and that tachycardia in a patient with an organic heart lesion may be due, not to the lesion, but to anxiety concerning it

Ketosis and *anoxaemia* are other conditions which render the medullary centres more sensitive to accelerator and less sensitive to inhibitory reflexes, thereby increasing the sympathetic tone and producing tachycardia *Anaemias*, when of sufficient severity to cause anoxaemia of the medullary centres, give rise to tachycardia

(3) **Peripheral Stimulation of the Sympathetic.**—The most important cause of *tachycardia* in this group is *thyrotoxicosis*, the tachycardia is often considerable. *Drugs*, including adrenaline and thyroid extract, will also cause tachycardia Some women take thyroid surreptitiously for the

[illegible] may be

[illegible] inflammatory lesions causing tachycardia; while

activity. Some patients are aware of a "missed beat". On examination a beat is dropped both at the heart and at the radial pulse. This must be distinguished from a dropped beat *at the radial pulse due to an extrasystole*, recognised on auscultation by the occurrence of a premature heart beat, and much more frequent as a cause of "missed beats". *Incomplete auriculo-ventricular block also causes missed beats both* at the radial pulse and on auscultation. When due to sino-auricular block the missed beats usually cease to occur after exercise; with *incomplete auriculo-ventricular block* beats may be missed more frequently after exercise. In the cardiogram (Fig. 17, B) no P is seen in the long interval when due to sino-auricular block (cf Fig. 28, B which shows a missed beat due to auriculo-ventricular block; the second P wave, occurring in the long pause, has no QRS or T following it)

Simple Tachycardia

In simple (or sinus) tachycardia, stimuli are liberated from the sino-auricular node at a greater rate than usual. The final step in its production is always an increase in sympathetic tone with a diminution in vagal tone. There are many ways in which this can be brought about.

(1) **Reflex Tachycardia.**—Painful stimuli from almost any organ in the body can excite a reflex tachycardia. Apart from this, there are two vascular reflexes which affect the heart rate by altering the vago-sympathetic tone, they are the cardio-accelerator reflex, and the carotid sinus reflex.

The *cardio-accelerator reflex* is initiated by distension of the right auricle and results in reflex acceleration of the heart (Bainbridge). This is the mechanism whereby tachycardia occurs during *exercise* which leads to an increase in the venous return, hyperpnoea will also increase the venous return causing tachycardia. In *right heart failure* dilatation of the right auricle gives rise to a reflex tachycardia, provided the cardiac rhythm is normal.

The *carotid sinus reflex* is evoked by a change of pressure in the carotid sinus, the afferent nerve being the glosso-pharyngeal. A rise in carotid pressure induces a reflex increase in vagal tone with slowing of the heart, while a fall in pressure augments the sympathetic tone with acceleration of the heart.

A fall in the carotid pressure is the explanation of the tachycardia in *shock*, in *haemorrhage*, in conditions of *dehydration*, and in *left heart failure*.

(2) Central Stimulation of the Sympathetic Centres in the Medulla.—*Bacterial toxins* are frequently responsible for central stimulation of the sympathetic with resulting tachycardia; thus tachycardia is present in most *acute infective illnesses* (with a few exceptions such as typhoid), and in many *chronic infections* such as tuberculosis, chronic pyuria, etc. Tachycardia often persists for some time during *convalescence* from acute fevers, especially influenza; it would seem that stimulation of the centres during the acute stage has left them more sensitive to accelerator and less sensitive to inhibitory reflexes, a process which presumably corresponds to Sherrington's "facilitation of reflexes".

Emotion is another very common cause of tachycardia, perhaps the most frequent cause of all. The emotions are closely linked with the medullary centres subserving the vegetative nervous system, and they are capable of increasing either vagal or sympathetic tone, more often the latter. *Anxiety states* are very frequently responsible for a persistent tachycardia, and not infrequently for an erroneous diagnosis in consequence, it should be remembered that the presence of an organic cardiovascular lesion does not render the victim immune from anxiety, and that tachycardia in a patient with an organic heart lesion may be due, not to the lesion, but to anxiety concerning it.

Ketosis and *anoxaemia* are other conditions which render the medullary centres more sensitive to accelerator and less sensitive to inhibitory reflexes, thereby increasing the sympathetic tone and producing tachycardia. *Anaemias*, when of sufficient severity to cause anoxaemia of the medullary centres, give rise to tachycardia

(3) Peripheral Stimulation of the Sympathetic.—The most important cause of tachycardia in this group is *thyrotoxicosis*, the tachycardia is often considerable. *Drugs*, including adrenaline and thyroid extract, will also cause tachycardia. Some women take thyroid surreptitiously for slimming purposes, and thus is occasionally the explanation of an obscure tachycardia. It has been suggested that the sympathetic ganglia may be irritated by inflammatory lesions causing tachycardia; while

admitting the possibility, I have never yet seen any convincing evidence of this.

(4) **Paralysis of Vagus.**—This may be central in certain diseases of the medulla; I have seen it in progressive bulbar paralysis, in encephalitis lethargica, and after relief of pressure in cerebral abscess and cerebral tumour. Vagal paralysis can also be peripheral; it may occur in peripheral neuritis, as a sequel to tetanus, from pressure by tumours, or from the action of drugs of the belladonna group (atropine, belladonna, hyoscine, strammonium, xanthoxylum).

It should be noted that, while simple tachycardia is sometimes an indication that the heart is failing, and that a reflex *tachycardia can occur either in right or in left heart failure*, it is much more often due to some cause unconnected with the heart. As an isolated physical sign, it should lead to a search for an acute or chronic infection, for hyperthyroidism, or for evidence of an anxiety state, these being the most frequent causes. A diagnosis of heart disease should never be based on tachycardia without other evidence of the existence of heart disease; and a diagnosis of cardiac failure should never be made unless there is independent evidence of defective circulation such as has been described in Chapter 2. Simple tachycardia provides a problem for the average practitioner almost daily: I have therefore considered it worth discussing in detail.

Clinical Features.—The rate may be anywhere from 90 up to 180, a rate above 150 is uncommon, and is more likely to be due to paroxysmal tachycardia or auricular flutter. It rarely remains constant, but increases with exercise and diminishes with rest Changes are always gradual, being spread over several beats, never abrupt as in paroxysmal tachycardia or flutter. In cardiograms, P and QRST follow one another in normal sequence but at a more rapid rate (Fig. 18, A) The increase in rate is brought about chiefly at the expense of the diastolic interval which is greatly shortened. With heart rates of 130 and upwards, P occurs before the termination of the preceding T, and as the frequency increases the P and T waves gradually tend to become fused (Fig 18, B) With rates above 150 the P and T waves may be so closely fused as to be indistinguishable from one another, and the record may simulate a paroxysmal tachycardia; as a rule they

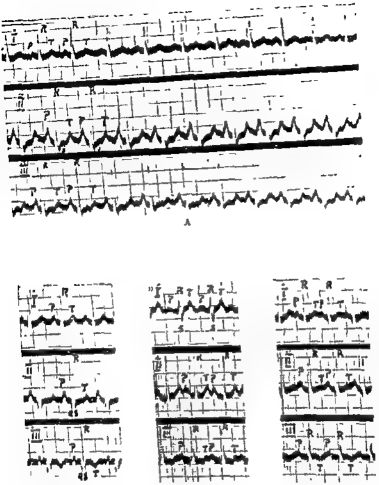


Fig [illegible] —Simple tachycardia

A *Simple tachycardia* Heart rate, 107 The diastolic interval is much shortened, but T and P are still distinct The individual complexes are normal

B

I

remain distinguishable in one lead, or failing this, a change in rate may cause their separation; but at times the distinction is impossible from a single record.

A rapidly beating heart in the absence of an increased circulation rate involves a smaller output per beat; the pulse is correspondingly weaker and the blood pressure lower. When *the tachycardia is associated with an increased circulation rate*, as is the case after exercise, or in thyrotoxic patients, the output per beat is normal or increased; the pulse is strong and the pulse pressure may be increased. With a normal circulation rate, tachycardia does not lower the cardiac reserve; many patients remain symptom-free, but in some the underlying anxiety state leads to complaint of breathlessness or palpitation on exertion ("effort syndrome"). With an increased circulation rate (thyrotoxicosis, anaemias, etc.) the cardiac reserve is diminished. If the coronary arteries are diseased, even in minor degree, the shortening of the diastolic interval resulting from tachycardia may interfere with their adequate filling, and the patient may have myocardial pain; but this never occurs with healthy coronary vessels. Tachycardia does not diminish the coronary blood flow in experimental animals despite the shortened diastole; the flow is actually increased during exercise in healthy hearts.

Treatment depends on seeking and removing the cause of the tachycardia. In differing circumstances this may involve *sedatives and reassurance for an anxiety state*, removal of sepsis or treatment of a chronic infection, treatment of thyrotoxicosis, or rest and digitalis for heart failure. The patient and his general health should be treated, never the tachycardia as such. *Digitalis and its allies are permissible only when the tachycardia is a manifestation of cardiac failure*; in all other cases they are not only valueless but often harmful, and they are therefore contra-indicated. Ergot and its alkaloid ergotamine (Femergin) appears to have a specific action on the sympathetic, and is valuable in combination with ammonium bromide in cases where tachycardia is due to an emotional state or to thyrotoxicosis. For tachycardia associated with fevers the proper treatment is to attempt to control the infection (sulphonamides, penicillin, etc.) coupled with tepid sponging when fever is high; digitalis has no place in the treatment of these cases.

Simple Bradycardia

Simple bradycardia is the exact reverse of simple tachycardia; it is due to increased activity of vagus with diminished activity of sympathetic. It is a common finding in *athletes*, [illegible] in the carotid artery which evokes the carotid sinus reflex. Bradycardia is characteristic of hypertension as long as the latter remains uncomplicated; though usually abolished when cardiac or renal failure supervene or when there is a complicating thyrotoxicosis or anxiety state, bradycardia occasionally persists despite cardiac failure, especially if the failure is of the congestive type. In a few individuals, painful stimuli excite a reflex bradycardia in place of the more usual tachycardia.

Central stimulation of the vagus is a frequent cause of bradycardia. In one group of cases it results from increased intracranial pressure, of which it is a well-known sign; thus it occurs with cerebral tumours, cerebral abscesses, depressed fracture of the skull, hydrocephalus, and in the *compression* stage of tuberculous *meningitis*. In certain individuals *emotion* affects the vagus rather than the sympathetic; this is exemplified by the popular expression, "His heart stood still with fear". Likewise *convalescence* in some patients is marked by bradycardia with low blood pressure and tendency to *faints* in place of the more usual tachycardia. The toxins of the *typhoid* group of organisms tend to affect the vagal rather than the sympathetic centres, and a relatively slow pulse is an important feature which aids the distinction between typhoid and many other fevers. Bradycardia is also frequent in *jaundice*.

Peripheral stimulation of the vagus provides the mechanism whereby certain *drugs* produce bradycardia. Physostigmine and pilocarpine act in this way, as does digitalis when it has any slowing effect at all on a normal cardiac rhythm.

Peripheral inhibition of the sympathetic will also cause bradycardia, and is presumably the explanation for the bradycardia of myxoedema. Ergotamine and ergotoxin can produce bradycardia by sympathetic paralysis. For anatomical reasons pressure is an unlikely cause of peripheral sympathetic paralysis.

Although heart failure tends to produce a reflex tachycardia, in hypothyroid patients and in hypertensive cases there are influences tending to produce bradycardia; in myxoedema,

bradycardia usually persists despite congestive failure, and in hypertensive cases it sometimes does so. In such cases thyroid extract is a valuable help in treatment of the cardiac failure; patients may, in fact, fail to improve until they receive thyroid as well as digitalis or diuretics. It should also be noted that, with the exception of hypothyroidism and hypertension, a simple bradycardia is never a sign of heart disease.

Clinical Features.—The pulse rate at rest is 60 or less, occasionally as low as 40; I have once seen a simple bradycardia with a pulse rate of 30 in a case of cerebral compression.

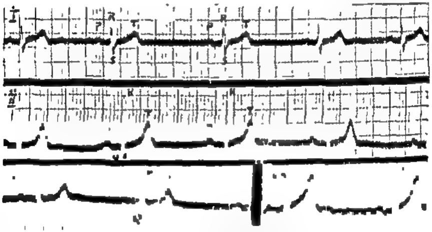


FIG. 19.—Simple bradycardia.

Simple bradycardia. Heart rate, 55.5. The diastolic intervals are much lengthened. The second portion of the lower strip is the chest lead (4R).

The rate does not remain constant; it is increased by exercise, though usually to a less extent than a normal heart rate would be. Changes in rate are always gradual, never abrupt. *Cardiograms* (Fig. 19) show beats spaced widely apart, with a long diastolic interval between T and the succeeding P.

Other conditions which cause a slow pulse and which must be distinguished from simple bradycardia are 2–1 heart block, complete heart block, and coupled extrasystoles. With coupled extrasystoles the sound of the extrasystole is heard on auscultation. In 2–1 heart block an extra sound, due to auricular contraction, is sometimes audible; the response of the pulse to exercise is less than in a simple bradycardia (e.g. a rise from 40 to 60 instead of to 80 or more). In complete heart

block the response to exercise is still smaller. Cardiograms readily distinguish the three conditions.

Treatment.—Apart from treating the cause (which includes the administration of thyroid extract in hypothyroid cases), simple bradycardia does not call for treatment.

EXTRASYSTOLES

These are the beats which arise from time to time in some situation other than the sino-auricular node ("*ectopic beats*"); they may arise in the auricle, in the auriculo-ventricular node or bundle, in the branches of the bundle, or in its terminal ramifications (Purkinje fibres) in the ventricles. According to their site of origin they are called auricular, nodal, septal, or ventricular. As a rule, only one kind of extrasystole occurs in any one patient; rarely, several different types occur in one patient and the significance is very different in these circumstances.

The extra beat is premature, that is, it occurs before a normal sinus beat is due. A stimulus liberated simultaneously with or just after a normal beat would encounter a *refractory* heart, and would simply fail to cause a contraction. Presumably in a patient with extrasystoles, some focus in the heart is more sensitive and so becomes capable of liberating stimuli independently; it is thought that the preceding beat excites the sensitive focus, causing it to commence building up its independent stimulus. In any given patient the time which elapses between the preceding beat and the extrasystole is constant, provided the extrasystoles are all arising from the same ectopic focus. If in a given case this interval is not constant, the case is one of interference dissociation or parasystole (see p. 117).

Aetiology.—Extrasystoles occur both in patients with healthy hearts and in persons with heart disease. They are very common in persons whose hearts show no other abnormality whatsoever; by themselves, therefore, they are not a sign of heart disease. As a rule, when they occur in a person with an organic heart lesion, they do not indicate that the heart disease is more serious than it would be in their absence—they are usually due to some extracardiac cause. There are, however, some exceptions to this rule, when a patient has

extrasystoles of more than one kind (arising from more than one focus) there is usually diffuse disease of the heart muscle; or they may be an indication of digitalis poisoning. Again, when extrasystoles occur in spite of a rapid heart rate, or when they are frequent immediately after exercise (instead of disappearing as they usually do), they suggest serious myocardial disease. In mitral stenosis frequent auricular extrasystoles often indicate that auricular fibrillation will soon develop.

Extrasystoles are more common in males, and are more frequent with advancing years. They are often due to some toxaemia (whether the patient has organic heart disease or not)—excessive tea, coffee, tobacco, alcohol, constipation, oral sepsis, etc. They often accompany gastro-intestinal disturbances—flatulent distension of the stomach. They are frequent in nervous patients, especially in response to fatigue, worry, or anxiety—and their effects are more noticed by patients who are fatigued or worried. They are frequent in women at the menopause. In all these circumstances they may disappear or become less frequent when the precipitating factor is found and treated. In patients with heart disease they are often produced by digitalis and they may be the first sign of its toxic action (though this does not necessarily mean that the digitalis must at once be stopped); extrasystoles produced by digitalis may arise from a single focus or from several different foci When extrasystoles occur in patients with heart disease they are particularly likely to arise in the chamber which is exposed to the extra strain—e g left auricle in mitral stenosis.

Clinical Features.—Extrasystoles occur before a normal beat *is expected, and are recognised clinically as premature* beats. If the premature beat occurs very early in diastole the heart contains little blood at the time the beat is a weak one and fails to open the aortic valve; the extrasystole produces a first sound, but no second sound, and no pulse wave If the premature beat occurs later in diastole the heart contains more blood; the beat is stronger and opens the aortic valve; the extrasystole produces a first and second sound, and sends a pulse wave to the wrist which is felt as a premature beat, though it is weaker than the normal beats because the filling of the heart was incomplete.

Unless the extrasystole is very premature, the heart is still contracting or refractory when the next sinus stimulus is due, and this stimulus therefore fails to produce a contraction; the extrasystole replaces the sinus beat. If the extrasystole has arisen in the ventricle it neither spreads back to the auricle nor disturbs the rhythm of the sinus; the auricle contracts in response to the sinus while the ventricle is still responding to the extrasystole. The next sinus beat develops at the expected time and causes a normal contraction of the whole heart. The pause which follows an extrasystole is therefore longer than that which follows a normal beat (post-extrasystolic pause); in the case of a ventricular extrasystole it is fully compensatory —the interval between the sinus beats preceding and following the extrasystole is exactly twice that between two successive sinus beats. An auricular extrasystole disturbs the stimulus which is brewing in the sinus node and the latter starts afresh to build up a stimulus, the interval which follows the extrasystole is slightly [illegible]
compensatory. It [illegible]
cular from auricul[illegible]
whether the post-extrasystolic pause is compensatory. In a patient with mitral stenosis an auricular extrasystole produces an auricular contraction and there is a presystolic murmur before its first sound; a ventricular extrasystole excites no auricular contraction and is not preceded by a presystolic murmur.

The interval which follows an extrasystole, being longer than a normal interval, allows of greater filling of the heart; the next sinus beat is therefore stronger than usual. It makes up for any slight deficiency in the circulation which might have resulted from the longer pause. Extrasystoles [illegible]

[illegible] the interval between sinus beats becomes shorter than the interval between a sinus beat and an extrasystole; the latter interval is constant in any given patient.

Extrasystoles may occur at long intervals or frequently. At times they recur regularly, for example, after every tenth, every fourth, or every third beat. Isolated extrasystoles produce either a dropped beat at the wrist, or a weak premature

beat followed by a long pause. Extrasystoles which alternate with sinus beats are called "coupled extrasystoles" and produce a "coupled pulse"; digitalis is a common though not invariable cause. Other possible causes of a coupled pulse are sinus coupling and 3–2 heart block; extrasystoles are by far the most common.

Many patients are unaware of extrasystoles—they have no abnormal sensations whatsoever. Some recognise the prematurity of the beat; some are aware of the long pause which follows the extrasystole; some are aware of the extra strong beat which follows it, which they feel as a thump in the chest. Any of these patients may complain of "palpitation", and if they can be induced to describe exactly what they feel, the diagnosis of extrasystoles can usually be made with certainty.

Occasionally extrasystoles are very frequent; occasionally they occur in short runs of two, three, or more. In such circumstances a bout of extrasystoles may be accompanied by a transient fall of blood pressure with faintness, dizziness, etc.

Diagnosis is generally easy from the typical sounds and the dropped or premature pulse beat. When coupled, extrasystoles may be mistaken for sinus coupling or 3–2 block. When frequent and irregular they may be mistaken for auricular fibrillation In most cases extrasystoles are abolished by exercise, whereas the irregularity of fibrillation becomes more evident. Extrasystoles which become more frequent after exercise or which persist despite tachycardia usually indicate serious myocardial disease or digitalis poisoning; in mitral stenosis frequent auricular extrasystoles may presage the onset of auricular fibrillation, in all other circumstances extrasystoles are innocent

Cardiograms *Auricular extrasystoles* show a premature and abnormal P wave—it differs from the P of the normal sinus beats and it may be inverted. The PR interval is often slightly longer than that of the sinus beats, but if the extrasystole arises close to the A–V node it is shorter. The ventricular complex which follows is usually normal, and is followed by a longer pause than usual (Fig. 20, A).

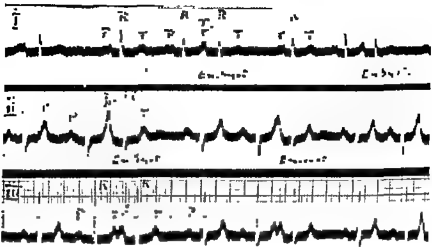
Nodal extrasystoles. The auricle and the ventricle contract simultaneously and the P wave is usually buried in QRS so as to be invisible. There is a premature QRS and T of normal type without any preceding P (Fig 20, C) At times, if the

extrasystole arises high in the A-V node, the auricle contracts a fraction of a second before the ventricle, and QRS is preceded by an inverted P with a very short PR interval (Fig. 20, B) In other cases, when the extrasystole arises lower down, in the main stem of the bundle, the ventricle contracts a fraction of a second before the auricle, and the inverted P follows QRS—it can be seen in the RT interval.

Ventricular extrasystoles. These are characterised by a premature and abnormal ventricular complex, it is broadened and notched, and with initial and terminal deflections directed in opposite directions When the initial deflection is downward in lead 1 and upward in lead 3, the extrasystole is usually left ventricular, when upwards in lead 1 and downwards in lead 3, it is usually right ventricular (Fig. 20, D and E). The position of the heart affects the direction of the deflections in the limb leads, the site of origin can be determined from chest leads, the initial deflection being upright when the chest electrode is over it, but downwards when the chest electrode is elsewhere.

Treatment of Extrasystoles.—This is a matter of determining the cause if possible and treating that. When extrasystoles occur in patients with heart disease the treatment is that of the underlying cardiac condition or of any heart failure present. The presence of extrasystoles does not contra-indicate the use of digitalis if otherwise indicated, but it should be remembered that the appearance of extrasystoles may be an early indication of the toxic action of digitalis

When extrasystoles are accidentally found in a person who is free from heart disease, and when they are causing no subjective symptoms, they should be ignored; any state of general ill-health should be corrected If the [illegible] patients a mild sedative such as phenobarbitone or ammonium bromide will often relieve the discomfort When associated with flatulence, kaolin or charcoal will often give symptomatic relief while suitable dietetic measures are instituted Quinidine sulphate in doses of 3 to 6 grains thrice daily will prevent extrasystoles temporarily, but it will not affect the underlying state of ill-health, and the extrasystoles usually return as soon as the drug is discontinued, in



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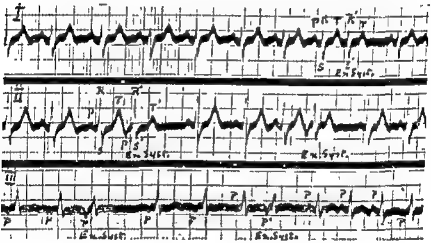


B

FIG 20 —Extrasystoles

A *Auricular extrasystoles* The premature P wave is superimposed on T, increasing its height in leads 1 and 2 (compare the second T in lead 2 with the first and third) In lead 3, P and the preceding T are only partially fused, giving rise to a bifid wave

B *Nodal extrasystoles (arising in upper part of node)* The auricle is stimulated in a reversed direction and contracts just before the ventricle, the PR interval of the extrasystole is [illegible]

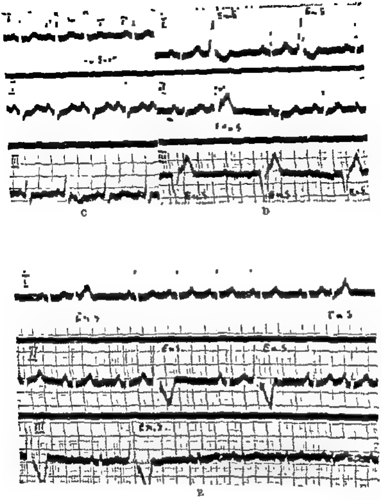


FIG. 20.—Extrasystoles (contd.)

C

D. *Ventricular extrasystoles.* Type with initial deflection upwards in lead 1 and downwards in lead 3. Commonly though not invariably right basal. There is left axial deviation with small QRS 1 and inverted T3.

E. *Ventricular extrasystoles.* Type with initial deflection downwards in lead 1 and upwards in lead 3. Commonly though not invariably left apical.

isolated cases it may be useful as a temporary palliative while other suitable therapeutic measures are being undertaken.

Nodal Escape and Nodal Rhythm

If, for any reason, the rate of the sino-auricular node becomes slowed to such an extent that it falls below the automatic rate

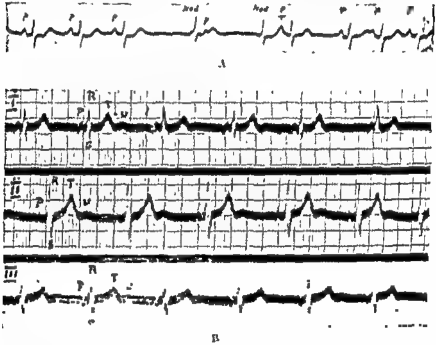


Fig 21 —Nodal escape and nodal rhythm

A *Nodal escape* The auricles are responding to the sino-auricular node and are showing a well-marked sinus arrhythmia The first three P waves are followed by ventricular [illegible] this is a [illegible] increases again

B *Nodal rhythm* The P waves are low and diphasic The PR interval is short (0 12 sec) The heart rate is 70 A small U can be seen in each lead

of the auriculo-ventricular node, the latter will liberate a stimulus before the auricles have contracted. This stimulus excites a ventricular contraction , it may or may not spread back to the auricle exciting a retrograde contraction. In the former case both auricles and ventricles contract to the auriculo-ventricular nodal stimulus, the contraction in the auricles spreading in the reverse direction and giving rise to

an inverted P wave. In the latter case the ventricles contract to the auriculo-ventricular nodal stimulus while the auricles contract a fraction of a second later to the delayed sino-auricular stimulus In either case the auriculo-ventricular node controls the ventricular rhythm until the sino-auricular rate increases and becomes greater than the automatic auriculo-ventricular rate. This phenomenon is known as "Nodal Escape" (see Fig. 21, A). In those cases where the auriculo-ventricular node controls the rhythm of both auricles and ventricles, the resulting rhythm is termed "Auriculo-ventricular Nodal Rhythm", or simply "Nodal Rhythm" (Fig. 21, B). Where the auricles continue to respond to the sino-auricular node while the ventricles respond to the auriculo-ventricular node, the term "Parasystole" is used, to imply that the heart is responding to two independent pace-makers (Fig. 21, A).

Nodal rhythm may occur in any condition in which considerable sinus slowing takes place, for example with extreme sinus arrhythmia, or with simple bradycardia. As a rule, when the sino-auricular node is slowed by vagal activity, the vagus also exerts sufficient influence on the auriculo-ventricular node to prevent nodal escape; when this occurs it would seem to imply selective activity of the vagus Nodal rhythm is not necessarily a sign of heart disease but may occur either in healthy or diseased hearts. It usually disappears as soon as vagal activity is lessened, e g. during exercise, and therefore has no deleterious effect on the circulatory responses to exercise, posture, or other bodily activities. A nodal rhythm which persists irrespective of exercise, etc., is likely to impair the efficiency of the circulation

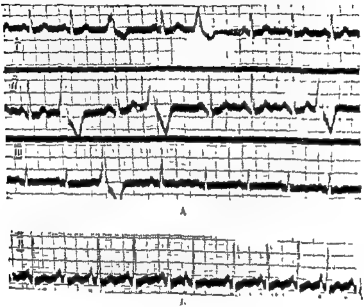
Parasystole and Interference Dissociation

The term "Parasystole" in its widest sense implies that the heart is responding to two independent pacemakers. One example has been described above in connection with nodal escape, another example is the state of affairs in complete heart block, in which the auricles respond to the sino-auricular node while the ventricles are controlled by a centre in the bundle below the site of the block. Theoretically, in cases of nodal escape with parasystole, it is necessary to postulate a "functional block" which prevents stimulation of the auricles

by the auriculo-ventricular node. Similarly with other varieties of parasystole where no organic block exists, a functional block must be postulated in order to explain the failure of the whole heart to respond to that centre which is the faster of the two. Nothing is known of the nature of this functional block nor as to its causation. It may occur in hearts which show no abnormality other than the resultant rhythm.

Occasionally it happens that two independent centres liberate stimuli rhythmically at different rates; the whole heart will respond to either stimulus, provided it is not refractory. The centre with the more rapid rate (often the sino-auricular node) controls the majority of the heart-beats, many of the stimuli produced by the second centre occurring at a time when the heart is either contracting or refractory. From time *to time, however, a stimulus is liberated from the ectopic centre* during a non-refractory phase and the heart responds to that stimulus which appears as an isolated extrasystole; that is to say, there is "escape" of the ectopic focus from time to time for a single beat. The ectopic focus may be in the auricle, in the auriculo-ventricular node, in the auriculo-ventricular bundle, or in its ramifications. The cardiogram at first sight might be mistaken for one showing simple extrasystoles (Fig. 22, A and 22, B). When these apparent extrasystoles are examined closely, it is seen that the interval between them and the preceding beat is not constant; the ectopic beat occurs sometimes early, sometimes late, in diastole. When the intervals between successive ectopic beats are measured, it is found that they are all exact multiples of a particular period which represents the automatic rate of the ectopic focus. If, starting from an ectopic beat, these intervals are measured off, it will be found that some ectopic stimuli fail to excite responses because they occur when the heart is in systole or refractory; as soon as a stimulus falls in a non-refractory phase, it excites a response. In this case, too, it is necessary to postulate a "functional block" which protects the ectopic focus from the beats of the dominant series, possibly the block may be organic. The condition is a rare one, both the examples illustrated here are from hypertensive patients with evidence of myocardial disease; a third example occurred in a man of 35 who had bronchitis and psychoneurotic symptoms but no evidence of organic heart disease.

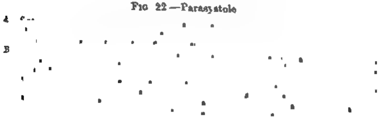
In cases of simple parasystole, as already explained, each centre is assumed to be protected from stimulation by the other ("protective block"), so that its rhythm remains undisturbed. Occasionally it happens that the dominant (more rapid) centre is not so protected. When the slower centre escapes, the



A

B

FIG 22.—Parasystole



resulting beat "explodes" the stimulus maturing in the dominant centre, which starts afresh to build up a stimulus. Every time an ectopic beat occurs it interferes with the basic rhythm of the dominant centre. This variety of parasystole is called "interference dissociation".

section)

In most cases of supraventricular tachycardia the ectopic focus controls the entire cardiac rhythm; but in many cases of ventricular tachycardia the auricles are protected from the ectopic focus by a retrograde block; the auricles contract in response to the sino-auricular node, while the ventricles respond at a considerably greater rate to a focus in the bundle or its branches. This provides yet another example of "parasystole".

Paroxysmal Tachycardia

Paroxysmal tachycardia is simply a succession of extrasystoles following one another rapidly, so that they completely replace the ordinary heart rhythm Extrasystoles sometimes occur in short runs of two, three, or more (Fig 23, A) There is no hard-and-fast line between these and paroxysmal tachycardia; the paroxysm may last for seconds, for minutes, for hours, or for days. While it lasts the cardiac rhythm is originating from some abnormal focus, in the auricle, in the A–V node, or in the ventricle, and it is spoken of as paroxysmal auricular, nodal, or ventricular tachycardia. It is often impossible to distinguish auricular from nodal tachycardia in a single cardiogram, the distinction does not appear to be a matter of practical importance, and they are conveniently considered together under the title of "supraventricular tachycardia" The rhythm is regular during the paroxysm, and the rate is between 125 and 240, very often in the neighbourhood of 200, so that the heart is rapid and regular.

The view regarding the nature and mechanism of paroxysmal tachycardia here expressed is that commonly accepted in this country In certain American and Continental circles an entirely different mechanism is thought to exist The alternative view postulates, in addition to the auriculo-ventricular Bundle of His, a second conducting bundle between auricles and ventricles, termed the Bundle of Kent. It is suggested that in supraventricular tachycardia a stimulus travelling down the Bundle of His excites ventricular contraction, this in turn excites a stimulus which travels up the Bundle of Kent, re-stimulating the auricles If the stimulus travels down via the Bundle of Kent and up via that of His, the ventricles are stimulated in an abnormal direction, and ventricular tachycardia results Thus, those who hold this view relate paroxysmal

tachycardia to auricular flutter and fibrillation rather than to extrasystoles, regarding it as an expression of "circus movement" They quote the occurrence of cardiograms with an abnormally short PR interval, yet a ventricular complex showing abnormal direction of stimulation of the ventricles (the "Short PR-Bundle-branch-block syndrome") as evidence of the existence of the Bundle of Kent, it is known that persons with this syndrome are liable to attacks of paroxysmal tachycardia. The evidence against the theory is, however, considerable; first, no satisfactory demonstration of the Bundle of Kent has been made anatomically in the human heart; secondly, paroxysmal tachycardia is common while the "short PR-BBB syndrome" is rare; and thirdly, it might be expected that, were this view correct, paroxysmal tachycardia would be familial with much greater frequency than is actually the case

Aetiology.—Paroxysmal supraventricular tachycardia (auricular or nodal) may occur in persons with healthy hearts, or in those with heart disease. Occasionally it is familial, as in the case of a woman of 70 whose paroxysms started at the age of 50 and who said that her mother suffered from identical attacks during the last twenty-five years of her life. More often there is no family history, but patients who suffer from attacks are often of a highly strung temperament In some 60 to 70 per cent of cases it is possible to obtain a history suggesting a reason for the initial attack, in the remainder the attack appears without apparent cause Excessive effort in the earlier days of convalescence from an acute illness, or sudden unaccustomed effort in a sedentary individual, will sometimes initiate the first attack Other factors to which first attacks appear to be related include pregnancy, the puerperium, and surgical operations, especially if any of these conditions are complicated by sepsis. The relationship of paroxysmal tachycardia to pregnancy is curious, some women have attacks only when they are pregnant; in others attacks cease during pregnancy to return after the puerperium, one of my patients developed paroxysmal tachycardia after her first uncomplicated confinement, continued to have very frequent attacks for two and a half years until she again became pregnant, then ceased to have attacks altogether, though it is now several years since the second pregnancy *Digitalis* is occasionally

responsible for inducing attacks, especially when it has been given in the absence of adequate indications.

Once the condition has made its appearance, it tends to recur at intervals throughout life. Subsequent attacks are often induced by emotion (anxiety), fatigue, gastro-intestinal disturbances, *excessive smoking, or by abuse of tea, coffee, or* alcohol; they are sometimes due to the ill-advised use of digitalis, occasionally to its justifiable therapeutic use. It is *my personal belief that the nervous factor is extremely important* in determining recurrences, and that recurrences would be less frequent if the medical profession did not regard them *as inevitable*, communicating a similar expectation and apprehension to their patients.

Paroxysmal ventricular tachycardia is on a rather different footing. It is in many cases a sequel to an attack of coronary thrombosis, and its development has an unfavourable bearing on the prognosis Occasionally it is a manifestation of digitalis poisoning in persons with serious organic heart disease, and again the prognosis is serious. Neubauer found three examples in children suffering from diphtheria, two of them died. It very rarely occurs in a person who is free from heart disease; Maurice Campbell has published one or two examples among a large series of cases of *paroxysmal tachycardia*.

Clinical Features.—The attack starts quite abruptly—often without discoverable exciting cause, but sometimes in response to fatigue, anxiety, etc. The duration varies from seconds or minutes up to hours or even a few days. the longest attack I have seen continued for three weeks, the shortest for a mere *four beats—the average is several hours* The patient may complain of palpitation with very rapid, regular heart action, and if so, he is aware of the sudden onset, and sometimes of *the abrupt termination of the paroxysm*, *but many* attacks are followed by extrasystoles in diminishing numbers, in which case the patient may say that they end gradually. Some have *cerebral anaemia during the attack with faintness*, dizziness, or tinnitus, occasionally they lose consciousness at the onset, there may be simple syncope or a Stokes-Adams seizure with a convulsion *In some cases the symptoms are* very slight and the attack passes almost unnoticed. As a rule there is little evidence of congestive failure, breathlessness and pulmonary or systemic venous *congestion only develop*

when the paroxysm occurs in an individual with organic heart disease, or if it is very prolonged When cerebral anaemia or venous congestion develop, they are probably attributable to inadequate filling of the heart in consequence of the shortened diastole, the cardiac output may drop as much as 33 per cent while the heart diminishes in size if there is any change at all.

During the attack the heart is rapid and regular, often between 180-220 per minute The pulse becomes very small and is usually uncountable, sometimes barely palpable; and some beats may be more easily felt than others, so that the pulse may seem irregular The onset of the paroxysm is quite abrupt, if observed (Fig 23, C), the end is equally abrupt and is followed by a long pause just like a single extrasystole (Fig 23, C) While it lasts the heart rate remains constant and is not altered by exercise. Pressure on the carotid artery will sometimes bring the attack to an end, but otherwise it does not affect the rate. Attacks can sometimes be ended by adopting some particular posture, by swallowing cold water, by inducing vomiting, etc., i e. by exciting a vagus reflex.

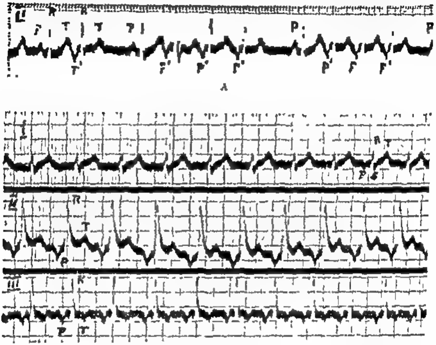
Diagnosis —In simple tachycardia the rate is not constant—it is increased further by exercise, etc., it rarely exceeds 160, though on occasions it may be up to 180, and changes in rate are gradual, not abrupt

Paroxysms of rapid regular heart action also occur in auricular flutter Here there may be sudden halving or doubling of the rate in response to exercise or rest, or to pressure on the carotid artery The rate is more likely to be 150-180 though rates up to 240 may occur Flutter is rare in the absence of organic heart disease, and attacks are more likely to be accompanied by signs and symptoms of congestive failure

Cardiograms In *supraventricular tachycardia* (Fig. 23, B, C D), P waves may or may not be visible. When visible, they are abnormal in shape, often inverted, and they occur in rapid regular series, each associated with a ventricular complex of normal shape Often, however, no P waves can be distinguished, they are buried in QRS or in the T of the preceding complex Distinction between auricular and nodal tachycardia is usually possible only if the onset of the paroxysm can be seen as in Fig 23, C, when the initial beat can be recognised as

auricular or nodal; or if the paroxysm is relatively slow as in Fig. 23, B, when the P waves are distinguishable.

Evans, using chest leads, has demonstrated auricular activity at twice the rate of the ventricles in a number of cases hitherto regarded as paroxysmal supraventricular tachycardia. He regards them as



B

FIG 23.—Paroxysmal supraventricular tachycardia.

A ... sinus beat and is followed by ... s a sinus beat and is fol- ... This could equally be ... eats are indicated by the

B

examples of auricular flutter with an unusually high flutter rate and a 2–1 block He goes so far as to imply that all cases of supraventricular tachycardia fall into this category, and speaks of the "unity of paroxysmal supraventricular tachycardia and auricular flutter" Campbell, working independently, has confirmed the findings in some cases and has shown that the 2–1 auriculo-ventricular ratio can often be detected even in the limb leads he adopts a non-committal attitude as to whether they are in reality cases of auri-

cular flutter or a special group of cases of paroxysmal supraventricular tachycardia. He has shown further that Evans's findings do

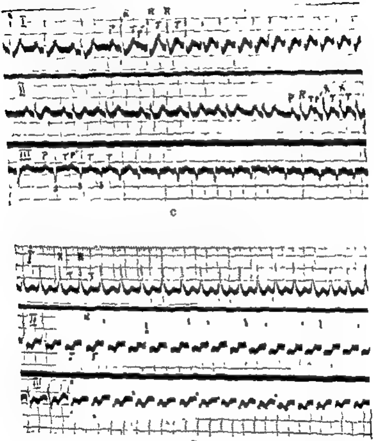


FIG. 23.—Paroxysmal supraventricular tachycardia (*contd.*)

C

D

not hold good for all cases of supraventricular tachycardia, some of which have a 1-1 auriculo-ventricular ratio, conforming to the

orthodox description. In the light of these recent findings, the relationship of paroxysmal supraventricular tachycardia to auricular flutter must be regarded as *sub judice*.

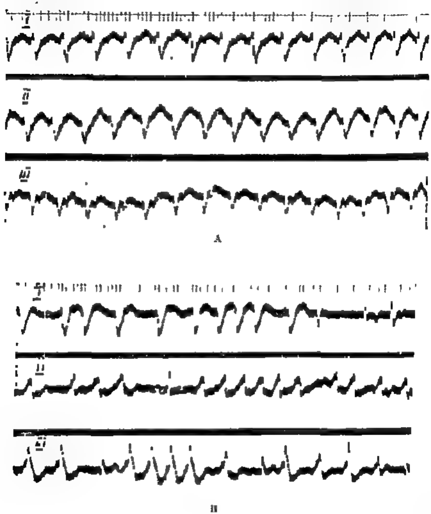


Fig 24 —Paroxysmal ventricular tachycardia

A *Paroxysmal ventricular tachycardia*
B *Short paroxysms of ventricular tachycardia*, occurring in the course of auricular fibrillation, isolated extrasystoles are also seen in this case, probably due to digitalis poisoning

In *ventricular tachycardia* there is a rapid series of abnormal ventricular complexes, each resembling the complex of a ventricular extrasystole. The auricular rhythm is sometimes undisturbed, P waves being visible here and there in the tracing (Fig. 24). In ventricular tachycardia the spacing of the beats

is sometimes not absolutely regular, though approximately so, the rate is often rather less than in supraventricular tachycardia, in many cases about 170.

Treatment of Paroxysmal Tachycardia.—When the patient is first seen during an attack, an attempt should be made to terminate it by pressure on the carotid artery against the transverse process of the 6th cervical vertebra, first on one side of the neck, and if this fails, on the other. Pressure on the eyeball, though painful, will occasionally succeed. Should these measures fail, the patient must be assured that the attack is not dangerous; death in a paroxysm is almost unknown. For many years quinidine has enjoyed an undeserved reputation for terminating attacks; it frequently fails, and when an attack does terminate while the patient is receiving quinidine it is likely that this is merely due to the natural tendency for the attack to end spontaneously. I have seen one patient in several prolonged attacks; the first one ceased while she was having quinidine; in the second, quinidine had no effect, but the attack subsided after a dose of "evipan," which induced sleep; the third ended while she was having "theominal" (a combination of theobromine with "luminal"), both quinidine and "evipan" having been ineffective; at the finish of her fourth attack she was having digitalis. If there is restlessness or anxiety with the attack, sedatives should be given. In the infrequent cases in which signs of cardiac failure appear, digitalis should be given. The occurrence of ketosis with an attack demands sodium bicarbonate and glucose; small doses of insulin (5 units) with the latter increases its efficacy.

In the intervals between attacks the patient's general health and habits should be reviewed, and corrected where necessary. Where there is no organic heart disease, any suggestion of invalidism should be scrupulously avoided; the patient should be assured that his heart is sound; provided his exercise tolerance and his general health are good he should have no restrictions placed on him; and finally, he should not be induced to expect further attacks, even though these may occur. Where the condition is complicating organic heart disease, restrictions depend on the nature and extent of the latter without regard to the attacks of paroxysmal tachycardia; the same general lines should be adopted in dealing with the patient's general health and habits.

Circus Movement, Auricular Fibrillation and Auricular Flutter

Mechanism.—On the Continent (German School) the mechanism of flutter and fibrillation is thought to be similar to that of paroxysmal tachycardia, viz. rapid formation of stimuli in an ectopic focus. In this country the mechanism is thought to be entirely different, namely a " circus movement ".

If a ring of heart muscle is cut from a tortoise heart and a stimulus is *applied at one point, it travels round the ring in* both directions till the two contractions meet at the opposite side of the ring , the stimuli cannot pass any further because they meet refractory muscle. But if, when the stimulus is applied, it is prevented from passing in one direction (e.g. by *momentarily applying a clamp), the stimulus* passes round in the other direction till it returns to the starting point ; and when it gets there, provided it has not travelled too rapidly, the refractory period has passed off, so that it continues to pass round the ring indefinitely.

Lewis has shown that something of this sort happens in auricular flutter and auricular fibrillation. It is thought that a ring of muscle in the auricle surrounding the orifices of the great veins acquires a diminished refractory period, so that if a stimulus once starts to pass round this ring in one direction, it continues to do so indefinitely At each point on its passage it stimulates the adjacent portion of the auricle (Fig. 25). At any particular moment some fibres of the auricle are contracting, but the auricle never contracts as a co-ordinate whole, and merely becomes passively dilated

Dilatation of the auricles is a characteristic pathological finding in cases of auricular fibrillation and flutter. The resultant slowing of the blood stream favours thrombosis, and thrombi are commonly found in the auricular appendages. Portions of thrombus may become dislodged, forming emboli, especially if the flutter or fibrillation ceases, so that the auricle contracts again. Infarcts are common in fibrillating or fluttering patients. If auricular fibrillation is directly observed in a dog's heart after opening the thorax, the contractions of individual fibres can be observed, also the fact that the auricle fails to contract as a whole

The rate at which the stimulus travels round the ring may be anywhere between 240 and 700 per minute. The slower

rates (240-400) are termed "flutter", while the more rapid rates (400-700) are termed "fibrillation". Between the two there is a zone which is intermediate, and cases are described as "impure flutter".

With each circuit one stimulus falls on the A-V node.

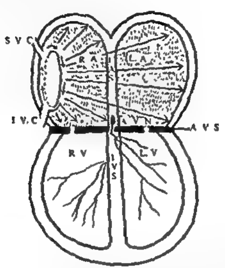


FIG 25.—Diagrammatic representation of circus movement The stimulus is assumed to travel round a circular path surrounding the orifices of the great veins as shown by the arrows At each point on its circuit the adjacent muscle fibres are stimulated as indicated by the linear arrows

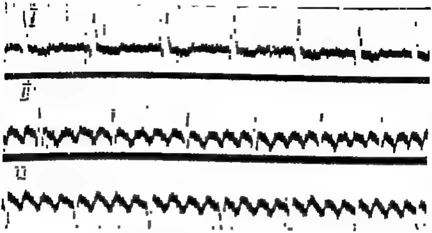
SVC = Superior vena cava	RV = Right ventricle
RA = Right auricle	IVS = Interventricular septum.
IAS = Interauricular septum	IVC Inferior vena cava
AVN = Auriculo-ventricular node continuous with the auriculo-ventricular bundle and its branches	LA Left auricle
	AVS = Auriculo-ventricular fibrous septum
	LV = Left ventricle

This transmits as many stimuli as it can to the ventricles, but the remainder are blocked

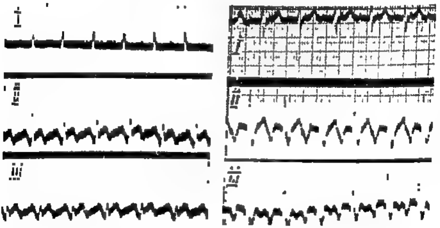
Auricular Flutter

Aetiology.—Flutter rarely occurs in the absence of serious organic heart disease. It may occur in almost any variety of heart disease—rheumatic, syphilitic, or arteriosclerotic—especially the latter. I have seen one case in a woman in whom I could demonstrate no gross cardiac lesion, the attack started

during a severe bout of sea-sickness, and it continued unabated despite various forms of treatment for twelve months, then it



A



B C

FIG 26 —Auricular flutter

A *Auricular flutter* Flutter rate, 312 4–1 block Ventricular rate, 78 Every fourth flutter wave is interrupted by a QRS complex in leads 2 and 3, the flutter wave is invisible in lead 1 The ventricular complexes show left axial deviation and inversion of T1
B *Auricular flutter* Flutter rate, 334 2–1 block Ventricular rate, 167
C

terminated spontaneously, only to recur again after an interval The reason for the change from normal rhythm to flutter is unknown.

Clinical Findings and Course.—With circus movement at a relatively slow rate and with a healthy conducting system, it

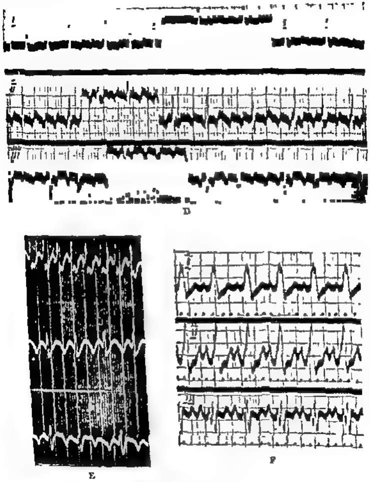


FIG. 26.—Auricular flutter (*contd.*)

D *Auricular flutter* Flutter rate, 333 Irregular block—varying between 2–

E

F *Auricular flutter* Flutter rate, 496 4–1 block Ventricular rate, 124 Two of the four flutter waves are distinctly seen, the third and fourth notch QRS, which is broadened (bundle-branch block) The peaks of the flutter waves have been indicated by *dots*

occasionally happens that all stimuli are conducted to the

during a severe bout of sea-sickness, and it continued unabated despite various forms of treatment for twelve months, then it

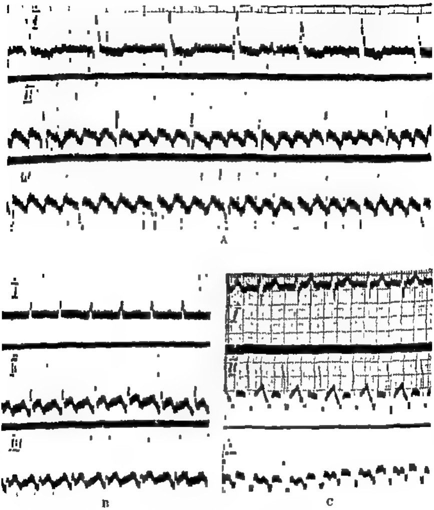


Fig 26 —Auricular flutter.

A ...ery fourth flutter ...ave is invisible in ...ion of T1

B

C ...17 Small flutter ...he second follow...es with S, which ...ised by a super-imposed a

terminated spontaneously, only to recur again after an interval. The reason for the change from normal rhythm to flutter is unknown.

terminate as cases of fibrillation. Generally speaking, the prognosis is poor.

Diagnosis.—Flutter is readily overlooked when the ventricular rate is between 70 and 120 and the rhythm is regular. The possibility of flutter should be borne in mind when the patient shows signs of congestive failure with a regular rhythm and relatively normal heart rate. The venous pulse in the neck shows only two waves (c and v) with each cycle instead of three (a, c, and v) which occur with a normal rhythm; sometimes it is possible to recognise this with the unaided eye. The occurrence of abrupt changes in the heart rate will also suggest the diagnosis. When the ventricular rate is rapid, flutter is likely to be mistaken for paroxysmal tachycardia; when the ventricular rhythm is irregular, the condition cannot be distinguished clinically from auricular fibrillation.

Cardiograms in flutter are very characteristic (Fig. 26). P waves have entirely disappeared, and their place is taken by a continuous wavy undulation of the base line (the flutter or "F" wave). Every second, third, or fourth of these is broken by a ventricular complex. T may be obscured or obvious; in the latter case the flutter wave is difficult to recognise at this point. Rarely every flutter wave is broken by a ventricular complex, and the cardiogram is difficult to distinguish from a paroxysmal tachycardia (Fig. 26, E).

Treatment.—Digitalis increases the grade of block and slows the ventricles; at the same time it usually increases the rate of the flutter wave converting the flutter to fibrillation; if the drug is discontinued at this point the circus movement may terminate and a normal rhythm may be restored. Quinidine has the reverse effect; it slows and ultimately abolishes the circus movement, after which one hopes the normal rhythm will be restored; while slowing the auricles it diminishes the degree of block and speeds up the ventricles, so that the patient's condition may be worse for a time. The general principles of treatment are similar to those which apply in auricular fibrillation (p. 138).

Auricular Fibrillation

Next to extrasystoles, auricular fibrillation is the most common cardiac irregularity. With rapid circus movement

ventricle, which therefore beats rapidly and regularly at about 240-250 per minute. With more rapid flutter rates every second or every third stimulus is conducted, and the ventricle beats regularly at a rate which is an exact fraction of the auricular rate. Thus, with a "flutter rate" of 300, the ventricle may beat at 150 (2–1 block), 100 (3–1 block), 75 (4–1 block), or 60 (5–1 block), etc. Exercise often causes an abrupt change in the degree of block, producing sudden halving or doubling of the rate, or a sudden increase or diminution by one-third or one-quarter; these abrupt changes in rate are characteristic of flutter, and the new rate bears an exact mathematical relationship to the old rate. With sudden doubling of the rate, attacks of regular tachycardia occur, which may be difficult to distinguish clinically from paroxysmal tachycardia; similar symptoms arise, but congestive failure is more often present.

Although the standard text-book description of flutter until recently was that the ventricular action is regular, in practice many patients with flutter have an irregular grade of block—varying 2–1, 3–1, and 4–1 block, so that now one, now two, now three stimuli may be blocked, and the ventricles are irregular. This is especially likely to be the case if the patient has been given digitalis; and in these circumstances the irregularity may be indistinguishable from that of fibrillation.

With slow ventricular action the circulation is often well maintained, though the fact that the auricles are out of action from the propulsive point of view impairs the patient's cardiac reserve. With more rapid ventricular action the pulse is small, the blood pressure falls, and venous congestion appears.

The onset of flutter is usually associated with a sudden deterioration in the patient's cardiac reserve, in many cases, especially those in which there is considerable cardiac enlargement and those in which the ventricular rate is rapid, symptoms and signs of congestive failure appear. In the earlier stages flutter is often transient, occurring in paroxysms which may last for a few days or a few weeks, the rhythm being normal in the interval. As time goes on, the paroxysms tend to become more prolonged and the intervals shorter, until the abnormal rhythm remains permanently established. There is also a tendency for flutter to pass into fibrillation in the course of time, so that some cases which have commenced as flutter

chronic heart disease and thyrotoxicosis in a case of fibrillation. Exceptionally, some toxaemia other than thyrotoxicosis may bring it on; for example, ketosis. In persons with an organic heart lesion which predisposes them to fibrillation, an acute infection, such as a cold, bronchitis, etc., may determine the onset; in others, excessive muscular exertion may do so. The common causes, in order of frequency, are (1) mitral stenosis, (2) thyrotoxicosis, (3) arteriosclerosis

Pathology.—The auricles are dilated and often contain thrombus in their appendages; infarcts are common in various organs. There are usually signs of congestive failure. In addition, the characteristic findings of the primary disease are present.

Symptoms.—The onset of the fibrillation is sudden, but the patient will only be aware of this if his circulation has hitherto been reasonably efficient He may complain of palpitation and describe the totally irregular heart action; he may have sudden breathlessness followed by rapid development of venous congestion; or he may experience faintness or syncope. On the other hand, if the heart is already digitalised, or if the bundle is defective as a result of disease, the onset of fibrillation may be associated with slowing of the ventricular rate and with improvement in his symptoms. In many cases the patient is unaware of the change in rhythm

The *physical signs* are those of the primary disease plus the characteristic total irregularity of heart sounds and pulse, the irregular blood pressure, and the pulse deficit

Diagnosis.—Fibrillation could be mistaken for frequent extrasystoles; exercise usually abolishes these, while it makes the irregularity of fibrillation more obvious. Flutter with irregular block usually cannot be distinguished clinically. Fibrillation with slow ventricular action is often overlooked, but can be detected if sufficient care is paid to the rhythm; if in doubt, the effect of exercise should be tried

Cardiograms show that the normal P waves have disappeared There may be undulations of the base line similar to those occurring in flutter, but more rapid, less regular, and smaller, known as the "f" waves; often the undulations are very small and visible only in parts of the tracing, or in one or two leads; sometimes they are invisible in all leads. The ventricular complexes occur at irregular intervals, either frequently or infrequently (Fig. 27).

and frequent stimuli falling on the A–V node (400-700 per minute) only a small fraction are conducted, and the degree of block is almost always irregular; in very rare cases it is regular (Fig. 27, D). The ventricles are, as a rule, quite irregular both in time and force. The rate depends on the degree of block, varying between 200 and 50 or less. The more rapid the rate the larger the proportion of weak beats, weak because the ventricles are incompletely filled; such beats are ineffective while wasteful of cardiac energy. They do not send a palpable pulse wave to the wrist; hence the pulse rate, as counted at the wrist, is less than the heart rate as counted with a stethoscope or by palpating the apex, the difference being known as the "pulse deficit"; with slow ventricular action, pulse rate and heart rate may be almost identical. Fibrillation with rapid ventricular action is often associated with congestive failure; with a moderate or normal ventricular rate it impairs the cardiac reserve; with a slow ventricular rate the circulatory reserve may remain good. The pulse deficit is thus quite a good guide to the degree of impairment of the circulation. Since the beats in fibrillation are constantly varying in force, the blood pressure varies from beat to beat, as the pressure in the sphygmomanometer is lowered, more and more beats come through. The variation in blood pressure from beat to beat is characteristic of fibrillation; the more rapid the heart rate the greater the variation, and vice versa, with relatively slow ventricular action the beats may all come through at nearly the same pressure.

Aetiology.—Auricular fibrillation very often occurs in grossly damaged hearts, but it is also found frequently in thyrotoxicosis where the heart seems undamaged except for the presence of the fibrillation. Rarely it occurs in an apparently healthy heart in the absence of thyrotoxicosis. It is more frequent in the chronic than in the acute stages of heart disease, and especially in those forms which throw additional strain on the auricles, thus the most common cause is mitral stenosis; it is infrequent in aortic valvular disease. It is fairly frequent in arteriosclerotic heart disease, but much less so in syphilitic or hypertensive heart disease. Rarely it develops temporarily during the acute stages of heart disease, for example, in rheumatic carditis, or soon after a coronary thrombosis. It is not uncommon to find a combination of

chronic heart disease and thyrotoxicosis in a case of fibrillation. Exceptionally, some toxaemia other than thyrotoxicosis may bring it on; for example, ketosis. In persons with an organic heart lesion which predisposes them to fibrillation, an acute infection, such as a cold, bronchitis, etc , may determine the onset; in others, excessive muscular exertion may do so. The common causes, in order of frequency, are (1) mitral stenosis, (2) thyrotoxicosis, (3) arteriosclerosis.

Pathology.—The auricles are dilated and often contain thrombus in their appendages; infarcts are common in various organs. There are usually signs of congestive failure. In addition, the characteristic findings of the primary disease are present.

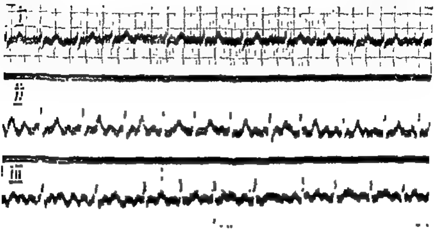
Symptoms.—The onset of the fibrillation is sudden, but the patient will only be aware of this if his circulation has hitherto been reasonably efficient. He may complain of palpitation and describe the totally irregular heart action, he may have sudden breathlessness followed by rapid development of venous congestion; or he may experience faintness or syncope On the other hand, if the heart is already digitalised, or if the bundle is defective as a result of disease, the onset of fibrillation may be associated with slowing of the ventricular rate and with improvement in his symptoms. In many cases the patient is unaware of the change in rhythm

The *physical signs* are those of the primary disease plus the characteristic total irregularity of heart sounds and pulse, the irregular blood pressure, and the pulse deficit

Diagnosis.—Fibrillation could be mistaken for frequent extrasystoles; exercise usually abolishes these, while it makes the irregularity of fibrillation more obvious. Flutter with irregular block usually cannot be distinguished clinically Fibrillation with slow ventricular action is often overlooked, but can be detected if sufficient care is paid to the rhythm, if in doubt, the effect of exercise should be tried.

Cardiograms show that the normal P waves have disappeared There may be undulations of the base line similar to those occurring in flutter, but more rapid less regular, and smaller, known as the "f" waves, often the undulations are very small and visible only in parts of the tracing, or in one or two leads; sometimes they are invisible in all leads. The ventricular complexes occur at irregular intervals, either frequently or infrequently (Fig 27)

The spiky irregularity of the base line produced by a dirty contact or by tremor of the patient's muscles should not be mistaken for the "f" waves of fibrillation; nor should the



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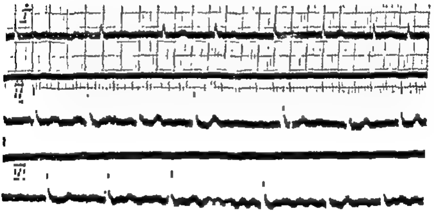


B

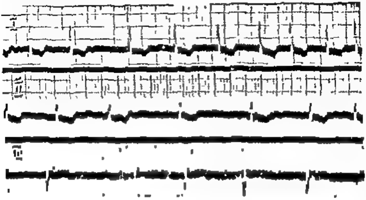
FIG 27.—Auricular fibrillation

A *Auricular fibrillation* The fibrillation wave is coarse in leads 2 and 3 The rate of circus movement is about 500 The ventricular beats are rapid and irregular, rate about 125 The ventricular complexes show slight right axial deviation. (From a case of mitral stenosis)

B. *Auricular fibrillation* In places the fibrillation wave is easily seen, in others it is invisible The fibrillation rate is 428 The ventricles are totally irregular, but their rate is normal (75 to 80)

regular spiky tremor produced by interference from alternating current lighting—its speed corresponds to 25 or 50 cycles per second, as the case may be.

Course.—Auricular fibrillation may occur in paroxysms when it first develops, or it may be permanent from the start.



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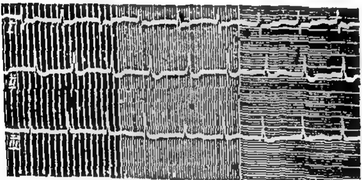

D

Fig 27.—Auricular fibrillation (*contd*)

When paroxysmal, the interval *between* paroxysms tends to

shorten while their duration tends to lengthen, and ultimately the condition becomes established and permanent. Sooner or later congestive failure develops, or a fatal termination may be brought about by an infarct

Treatment.—(1) If the fibrillation is secondary to thyrotoxicosis, it is essential to treat the thyrotoxicosis; and when this is successfully accomplished, the fibrillation will stop spontaneously *in most cases* *If it is thought that the case* is one of the rare ones in which the fibrillation is secondary to some other toxaemia, that should likewise be treated.

(2) *When secondary to an acute heart lesion such as active* rheumatic carditis or recent coronary thrombosis, the patient should be treated on the usual lines for these (rest, etc.), and the fibrillation will often disappear spontaneously. On no account should *quinidine* be used in these cases, and digitalis should be used only if there is congestive failure.

(3) In the remaining cases, where fibrillation is secondary to chronic heart disease, there are three possible courses:

(*a*) *If the ventricular rate is spontaneously slow, if there* is no congestive failure and fairly good exercise tolerance, it is often best to *leave the fibrillation alone.* Digitalis is not required All that is necessary is to restrict exercise to what the heart will stand without undue breathlessness *Before attempting* to restore normal rhythm with quinidine, one should be sure that it is safe to do so, and that the patient will be better off, not worse off, with a normal rhythm

(b) *Digitalis control.* If the ventricular action is rapid, if there are signs of congestive failure, or if the exercise tolerance is much impaired, an attempt should be made to control the ventricular rate with digitalis While there is evidence of congestive failure, the patient remains in bed Digitalis is given in full doses—e g 15-20 minims of the tincture or 1½ grains of digitalis leaf, four-hourly, until the heart rate drops to about 80. It is then stopped, and re-started in smaller dosage until the amount required to keep the heart rate at 80 is found. The patient is kept on this dose, but it may be necessary to intermit the treatment, that is, to leave the patient without digitalis for a few days from time to time if nausea, extrasystoles, or coupling develop An alternative method for the initial dosage ("Massive dosage") is to calculate the total amount required, allow 0 15 c.c. of standard-

ised tincture per pound of body weight excluding oedema (i e. base the calculation on the patient's weight before he became oedematous), subtract from this any digitalis which may have been given in the previous three days; and give the calculated amount either in one dose or in three doses at 6-hourly intervals The total dose usually works out at between 4 and 8 drams; no further digitalis is required for several days, after which a maintenance dose is instituted I have not seen any advantage for the massive dose method over the usual method In urgent cases strophanthin ($\frac{1}{100}$ to $\frac{1}{50}$ gr.) or digoxin 1 mg may be given intravenously, instead of digitalis Digitalis acts by slowing A-V conduction, slowing the ventricles, and cutting out the weak ineffective beats; it does not stop the fibrillation, in fact it tends to perpetuate it. Digitalis control is the method of choice for most cases.

(c) *Quinidine treatment* Quinidine acts by increasing the refractory period of the auricle, until a point is reached at which the circus movement is brought to an end, the whole heart stops for a brief period and then starts again, in many cases with a normal rhythm, but in some the fibrillation is resumed, occasionally the heart fails to start again, and the patient dies suddenly during the treatment. This is especially likely in the fibrillation of active cardiac disease (active carditis, recent coronary thrombosis), and quinidine is absolutely contra-indicated in these circumstances It is also contra-indicated in long-standing fibrillation, where there is probably thrombus in the auricles, and a probability of embolism if the normal rhythm is restored, it is absolutely contra-indicated in patients with a history of former embolism In the earlier stages of treatment the ventricular rate is increased and any symptoms of congestive failure are aggravated; quinidine is therefore contra-indicated in the presence of congestive failure till this has been controlled by rest and digitalis When these contra-indications are considered, it will be seen that comparatively few cases remain which are suitable for quinidine and before treating them with quinidine, one must satisfy oneself that the patient will be better off with a normal rhythm than with the fibrillation, remembering that a fibrillating heart can be controlled by digitalis while a regular rhythm cannot There remain a very few cases in which quinidine is indicated and justified, it is particularly useful in a thyrotoxic patient in

whom fibrillation persists after the thyrotoxicosis has been adequately treated by operation or thiouracil ; in an occasional patient with quiescent mitral stenosis in whom fibrillation has followed a specific act of over-exertion and whose symptoms *indicate that his reserve is worse while fibrillating* ; or for a case in which fibrillation has followed an acute infection or has arisen during acute heart disease and has not disappeared during convalescence.

Before starting the treatment proper, the patient should be given a test dose of 3 grains to detect idiosyncrasy. If there are no untoward results, he is given 6 grains next day in one dose, then two, three, four, and five doses on successive days , next day he receives a dose of 12 with four of 6 grains. Some give the doses at two-hourly intervals, some spread them over the 24 hours. The course is stopped when regular rhythm is restored, or at the end of the seventh day, whichever occurs first. After restoration of regular rhythm small doses (3 grains) should be given thrice daily for a few days to prevent recurrence.

In patients with paroxysmal fibrillation, small doses of quinidine may be used as a preventative treatment.

TABLE OF QUINIDINE DOSAGE

Time	Day						
	1	2	3	4	5	6	7
6 a m	6	6	6	6	6	6	6
8 a m.	—	6	6	6	6	12	12
10 a m	—	—	6	6	6	6	6
12 noon	—	—	—	6	6	6	6
2 p m	—	—	—	—	6	6	6

Slightly modified from Campbell and Gordon (1936).

Heart Block

Heart block is a condition in which there is a disturbance in conduction between the auricles and the ventricles. It may be congenital or acquired.

Congenital heart block is due to absence of a portion of the bundle. *It is often associated with a defect in the interventricular septum*, but this is not invariably present. The block is always complete. The ventricular rate is often rather higher than in acquired complete block, viz. 40-45 as compared with 30-35 in acquired cases.

Acquired heart block may be organic, toxic, or nervous in origin. *Organic block* results when any type of lesion affects the A–V bundle. In the acute stages of rheumatic carditis minor grades of block are common; in the chronic stages block is less frequent, but any grade may occur. Block also occurs in a proportion of cases of syphilitic heart disease, and occasionally in diphtheria. Coronary disease is a relatively frequent cause of block. *Toxic block* is produced by digitalis, which has a specific effect in depressing the conductivity of the bundle and is, in fact, used in the treatment of auricular fibrillation for this very purpose. *Nervous block* is due to activity of the vagus. In a healthy heart, vagal activity may cause slight slowing of conduction, but rarely produces any greater grade of block than this. In a person whose conduction is already impaired by disease, activity of the vagus may greatly increase the grade of block; thus it may convert a latent into a partial, or a partial into a complete block.

A minute organic lesion is sufficient to produce block if situated in the right place; a much larger lesion of identical type, but situated elsewhere, causes no block. The significance of heart block depends on the circumstances in which it is found; if there is but one small lesion, the remainder of the myocardium being healthy, the block may do little harm and the prognosis is good; if the lesion causing the block is one of many scattered throughout the heart muscle, the condition is serious and the prognosis poor. The nature of the lesion also influences the prognosis, in acute rheumatic carditis block is often transient; in the acute stage of diphtheria death is frequent, but complete recovery is the rule if the patient can be tided over the danger period, on the other hand in [illegible] disease the [illegible]

h [illegible] delay in the passage of stimuli from auricles to ventricles, all beats are still conducted, but the PR interval in the cardiogram is lengthened beyond [illegible] 20 second (Fig. 28, A). In this stage the condition can only be diagnosed by cardiograms or polygrams, except it happens to occur in a patient with mitral stenosis, when the presystolic murmur becomes separated by a short interval from the first sound,

whom fibrillation persists after the thyrotoxicosis has been adequately treated by operation or thiouracil; in an occasional patient with quiescent mitral stenosis in whom fibrillation has followed a specific act of over-exertion and whose symptoms indicate that his reserve is worse while fibrillating; or for a case in which fibrillation has followed an acute infection or has arisen during acute heart disease and has not disappeared during convalescence.

Before starting the treatment proper, the patient should be given a test dose of 3 grains to detect idiosyncrasy. If there are no untoward results, he is given 6 grains next day in one dose, then two, three, four, and five doses on successive days; next day he receives a dose of 12 with four of 6 grains. Some give the doses at two-hourly intervals, some spread them over the 24 hours. The course is stopped when regular rhythm is restored, or at the end of the seventh day, whichever occurs first. After restoration of regular rhythm small doses (3 grains) should be *given thrice daily for a few days to prevent recurrence.*

In patients with paroxysmal fibrillation, small doses of quinidine may be used as a preventative treatment.

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10 a.m	—	—	6	6	6	6	6
12 noon	—	—	—	6	6	6	6
2 p.m	—	—	—	—	6	6	6

Slightly modified from Campbell and Gordon (1930)

Heart Block

Heart block is a condition in which there is a disturbance in conduction between the auricles and the ventricles. It may be congenital or acquired

Congenital heart block is due to absence of a portion of the bundle. It is often associated with a defect in the interventricular septum, but this is not invariably present. The block is always complete. The ventricular rate is often rather higher than in acquired *complete block, viz. 40-45 as compared with* 30-35 in acquired cases.

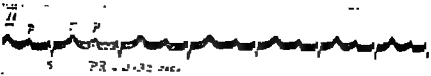
i e. becomes mid diastolic. The development of latent block is usually associated with an alteration in the quality of the first sound, which becomes softer.

Incomplete block. The next stage in the development of block occurs when occasional auricular beats fail to be conducted to the ventricles. Now and again a beat is "dropped" both at the heart and in the radial pulse. The dropped beats of extrasystoles can be differentiated by listening and recognising the sound of the extrasystole corresponding to the dropped beat The dropped beats of sino-auricular block will often disappear after effort; while in incomplete block beats tend to be dropped more frequently after exercise if the block is due to organic disease, though they may disappear after exercise if the block is in part due to vagus activity.

In one group of cases (Fig. 28, B) the cardiogram shows a gradual lengthening of the PR intervals in successive cycles until one stimulus fails to be conducted (Type A block—"Wenckebach periods"), these cases are always due to organic disease After the dropped beat the PR interval is short, but gradually lengthens again. In a second group of cases there is no such lengthening of the PR interval, every now and again a P wave is not followed by any QRS-T (Type B block) these cases may be organic or vagal.

As the capacity of the bundle to conduct becomes less, beats are dropped with increasing frequency till every fourth, every third, or every second beat fails to reach the ventricles (one in four or 4–3 block, one in three or 3–2 block, 2–1 block); with further impairment only every third, fourth, or fifth beat is conducted (3–1, 4–1, or 5–1 block), and so on Finally,

Fig 28.—Heart block.



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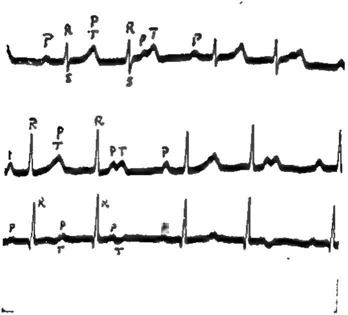

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D

E

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As the capacity of the bundle to conduct becomes less, beats are dropped with increasing frequency till every fourth, every third, or every second beat fails to reach the ventricles (one in four or 4-3 block, one in three or 3-2 block, 2-1 block); with further impairment only every third, fourth, or fifth beat is conducted (3-1, 4-1, or 5-1 block); and so on Finally,

Fig 28 —Heart block.

when there is complete interruption of conduction, the condition is called complete block.

3–2 *block*. Every third beat fails to reach the ventricles, so that one beat in three is dropped. The heart beats occur in pairs, separated by a long pause—i.e. the cardiac rhythm and pulse are coupled. It may be difficult to distinguish this clinically from coupled extrasystoles, but exercise may help (Fig. 28, C).

2–1 *block*. Every second beat is blocked. The ventricles beat regularly, at half the rate of the auricles—usually about 35-45, but sometimes higher if the auricles are beating more rapidly The heart rate is still increased by exercise, but only to half the extent that it would be with a normal rhythm. The blood pressure may be altered, the systolic tends to be higher and the diastolic lower, so that a high pulse pressure similar to that of aortic regurgitation occurs (e.g. 140/70). Despite the slow rate the circulation is usually well maintained, and the patient may have no symptoms of cardiac insufficiency. Cardiograms show two P waves for each QRS and T, one is followed by ventricular complexes, the second is not (Fig. 28, D)

Higher grades of block are associated with a still lower pulse rate. The patient may complain of symptoms of cerebral anaemia, such as faintness, dizziness, etc If the grade of block suddenly increases or if a partial block suddenly becomes complete, he may have a Stokes-Adams attack.

Complete block When block becomes complete, a centre in the A–V bundle below the seat of the block becomes active and controls the rhythm of the ventricles. These beat slowly and regularly—usually at a rate of 30-35 in acquired cases, and 40-45 in congenital cases. The auricles continue to beat independently at a normal rate The pulse is slow and regular, and is only slightly affected by exercise, it may rise two or three beats per minute but little more The systolic blood pressure tends to be raised and the diastolic to be lowered. Often there is little disturbance in the circulation; indeed the block causes none, and it is only when lesions similar to that causing the block are widespread throughout the myocardium that the patient has an impaired tolerance or congestive failure. Cardiograms show a regular series of P waves at one rate, and a regular series of QRS and T waves at a different and slower

rate (Fig 28, E); the two rates are not mathematically related. At times a P wave may occur just before a ventricular complex, but this is accidental; the next P probably coincides with T and notches it, the next may be buried in the following QRS, and so on, in fact all possible relationship of P to QRS and T will be found if a sufficient length of tracing is examined.

Symptoms.—In many cases heart block causes no symptoms; in others the symptoms, if any, are due to the underlying heart disease Occasionally there are symptoms due directly to the heart block These may take the form of attacks of faintness or dizziness, but the most characteristic syndrome is the Stokes-Adams attack In these attacks, which usually come on abruptly and without warning, the patient suddenly loses consciousness, he is usually deadly pale when the attack comes on, though I have seen one case in which colour remained unchanged throughout. After a few seconds convulsive twitchings appear in the muscles, and this may be followed by a generalised epileptiform convulsion The pulse may be very slow (15, 20, or 25 per minute), or it may be abolished entirely for as long as a minute, during which period no heart sounds can be heard on auscultation After half a minute to a minute or thereby, the pulse returns or increases in rate, the twitchings cease and the patient regains consciousness.

Formerly it was taught that the Stokes Adams attack was associated with a sudden increase in the grade of heart block, for example if partial block suddenly becomes complete, it was assumed that a period of ventricular standstill occurred before idioventricular rhythm became established. While it cannot be denied that this is a possible mechanism, a number of cases have now been cardiographed during attacks and none showed this particular mechanism. The observed attacks have occurred without any alteration in the grade of block ... when block ... attack ... cular st ... systole) for periods up to one minute In another group cessation of pulse and heart sounds were shown to be associated with ventricular tachycardia or ventricular fibrillation. In a third group the attack was ushered in by ventricular tachycardia or ventricular fibrillation and the short paroxysm was followed by a period of ventricular standstill It was further shown that cases in which the

paroxysm was associated with either ventricular tachycardia or ventricular fibrillation carried a more serious prognosis; almost all the patients with this type were dead within a year. When the attacks were associated with ventricular standstill alone, the prognosis was considerably better, and the majority of the patients lived for two years or more.

Loss of consciousness with convulsive twitchings may occur at the onset of other abnormalities of rhythm, for example at the onset of auricular fibrillation or of an attack of paroxysmal tachycardia. The term "Stokes-Adams" attack, however, is usually restricted to those cases in which the condition is associated with heart block.

Treatment of Heart Block.—In the absence of Stokes-Adams attacks the patient is treated on general lines; thus if his exercise tolerance is good, he is allowed to lead a more or less normal life and to continue at work, provided the nature of his work is not such that the unexpected development of an attack would involve danger of accident to himself or others. Impaired cardiac reserve calls for appropriate restriction of activities. Cardiac failure is treated with rest, and digitalis is not contra-indicated if rest, alone or combined with diuretics, fails to abolish the oedema; but if digitalis is given, the patient needs even more careful watching than when it is used for other forms of heart disease (I have seen digitalis cause Stokes-Adams attacks when used for the treatment of auricular flutter.)

Until recently the treatment for a Stokes-Adams attack due to heart block was universally accepted to be adrenaline—given subcutaneously if the pulse was merely slow, or intraventricularly if the pulse was absent. The more recent findings regarding the mechanism of Stokes-Adams attacks cast grave doubts on the wisdom of this accepted treatment: if the attacks are associated with ventricular fibrillation or ventricular tachycardia, adrenaline is more likely to aggravate or perpetuate the condition than to cure it—and it seems possible that the routine use of adrenaline may be the explanation of the more serious prognosis in this group of cases. On the other hand, if the attack is due to ventricular standstill, it is clear that subcutaneous adrenaline cannot reach the heart until the latter commences to contract again, and that recovery from an attack after adrenaline has been given subcutaneously cannot

possibly be due to the adrenaline; it is possible that intraventricular injection might stimulate the ventricles to contract again, but questionable whether the procedure is justified in view of the better prognosis in this class of case. Where there are frequent Stokes-Adams attacks, occurring several times daily or every few minutes, and where they are due to ventricular stand-still, the use of adrenalin subcutaneously or of ephedrine by mouth may lead to cessation of the attacks.

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to Goldberger's or Wilson's neutral terminal ("V" leads"). The CR and CF leads are bipolar leads. The potential of the limb itself changes with each cardiac cycle and introduces an error, positive in the case of the CR leads, either negative or positive, depending on the position of the heart in the case of the CF leads. A committee of the British Cardiac Society has recommended that the use of the bipolar CR and CF leads should be discontinued, the unipolar V leads being used instead.

A uniform notation has been agreed for identifying chest leads. The following points on the chest wall are denoted by numbers: 1, fourth right intercostal space at sternal edge; 2, fourth left intercostal space at sternal edge; 3, midway between 2 and 4; 4, fifth left intercostal space in the mid-clavicular line; 5, 6, and 7, on the same level as 4 in the anterior axillary line (5), mid-axillary line (6), and posterior axillary line (7) respectively [illegible] right si[illegible] 8, imme[illegible] points sometimes used are S (over the spine at the level of point 4), E (over the ensiform cartilage), and RUA (right costal margin just to the right of the ensiform cartilage. Chest leads are identified by prefixing the letters CR, CF, or V to the number of the selected point on the chest wall, e.g. CR2, CF4, or V6. The chest leads frequently give large deflections, and it may be convenient, in order to obtain the entire deflection on a single strip of film, to standardise the instrument so that 1 millivolt = ½ cm.; the resulting record should be marked N/2.

Unipolar Limb Leads

The actual variations in potential in a single limb are recorded by placing the exploring electrode on the limb in question, the indifferent galvanometer lead being connected to Goldberger's or Wilson's central terminal. They are known as VR (right arm), VL (left arm), and VF (left leg). They are useful for determining the position of the heart within the thorax and for showing posterior and lateral myocardial infarcts. The deflections in unipolar limb leads are often small, and it is usually more convenient to use the "augmented

CHAPTER 6

ABNORMALITIES OF THE VENTRICULAR COMPLEXES IN CARDIOGRAMS

The standard limb leads suffice in the vast majority of cases for the study of cardiac rhythm. They often give valuable information regarding the presence or absence of myocardial disease; but in some instances their interpretation is open to fallacy, ambiguous or erroneous conclusions may be drawn. For detailed study of myocardial lesions several chest leads are required in addition to limb leads.

Unipolar and Bipolar Leads

A lead obtained by placing two electrodes on the body surface is a *bipolar lead*; it records the *difference* between the potentials of the two chosen points at each given moment. The standard limb leads are bipolar leads A *unipolar lead* is obtained if one electrode (the "exploring electrode") is placed on the body surface while the second (or "indifferent electrode") is maintained constantly at zero potential; it records the actual potential changes at the point on the body surface to which the exploring electrode is applied.

To obtain a terminal which remains constantly at zero potential, advantage is taken of the fact that the changes in potential of the right arm, left arm, and left leg resulting from cardiac activity neutralise each other so as to add up to zero. Each limb may be connected directly to the central terminal (Goldberger method); or it may be connected to the central terminal through a resistance of 5000 ohms (Wilson method) The Goldberger method is the simpler, and gives equally good results.

Chest Leads

With the cardiograph switched to lead I, the left arm lead is connected to an electrode placed on the chest wall; the right-arm lead is connected to an electrode placed on the right arm ("CR leads"), on the left leg ("CF leads"), or attached

it; R therefore tends to be taller in leads overlying the left ventricle than in those from the right ventricle. This tendency is accentuated if there is left ventricular hypertrophy and is reversed by right ventricular hypertrophy.

In the average normal heart, therefore, leads V1 and V2 (overlying the right ventricle and septum) show a small narrow R occurring early in the QRS complex, and followed by a deep S. The height of R increases progressively from lead V1 to V4, reaching its maximum in V4 or V5; at the same time it occupies a rather later position in the QRS complex, so that a small Q is often present in V4, and almost invariably in V5 and V6. Meantime S is becoming progressively smaller and more narrow, it is usually absent in V6 and V7.

The spread of the recovery process produces the T wave. In persons with normal hearts, T is always upright in leads V3 to V7 inclusive and usually in V2; in V1 and V8 it may be upright, flat or inverted. The m[illegible] use [illegible] but [illegible] ing [illegible] in which the error may be either positive or negative, are even more misleading.

T inversion is often the *direct* result of ventricular hypertrophy, being found in leads V1 and V2 with *right ventricular* hypertrophy, and in leads V5 to V7 with left ventricular hypertrophy. Inversion of T can also be produced by myocardial ischaemia which *interferes* with the recovery process (see Ventricular Gradient, p. 165). On the other hand, myocardial necrosis or fibrosis prevents the spread of excitation through some part of the myocardium, it abolishes or delays the inscription of R in leads from that area, causing changes in the form of the QRS deflection.

In the majority of normal cardiograms the RT interval is iso-electric. Rarely the recovery process begins in some parts of the myocardium before excitation is complete in others; the inscription of T then commences before the inscription of QRS is complete, and the T wave arises from the descending limb of R or ascending limb of S, the RT (or ST) interval lying above or below the iso-electric level. In such cases the displacement of RT is seldom greater than 1 to 2 mm, and it persists unchanged over periods of weeks or months, lead V1 is most often affected, though the displacement may appear in

unipolar limb leads" denoted aVR, aVL, and aVF. These are obtained by using the Goldberger method and omitting the connection between the central terminal and the limb to be recorded

The Interpretation of Unipolar Chest Leads

As soon as the stimulus to contraction reaches the Purkinje network, the corresponding ventricular cavity becomes negative; an electrode overlying that ventricle inscribes a Q deflection Excitation of the ventricle spreads through the myocardium from the endocardium outwards. The spread of the excitation wave is associated with development of a positive charge in the affected muscle, the maximum positivity being attained when the wave reaches the pericardial surface. The outward spread of excitation through that portion of the myocardium underlying the exploring electrode produces the upstroke of R Immediately the sub-pericardial muscle fibres have been excited the excitation wave dies out and the positive charge disappears, the electrode once more records the negativity of the underlying ventricular cavity, the tracing drops abruptly from the peak of R to the bottom of S, the downstroke being termed the "intrinsic deflection". Finally, when excitation is complete in all parts of the ventricular muscle the negativity of the ventricular cavity disappears and the tracing returns to the iso-electric line.

The first part of the ventricular muscle to be excited is the interventricular septum with the adjacent myocardium on the anterior and posterior surfaces and at the apex. In leads from these areas the early positivity from myocardial excitation may cancel or even exceed the negativity of the ventricular cavity, so that the initial event in the QRS complex is the development of R. The intrinsic deflection occurs early, so that QRS has the form of a narrow R followed by a rather broader S. In leads taken from areas which are excited later, the development of R is delayed; the QRS complex consists of Q, R, and S. In leads from the last portion of the myocardium to be excited the intrinsic deflection is the final event in the QRS complex which consists of a rather broad Q followed by a narrow R and no S. The thicker the myocardium the greater is the positive charge generated by spread of excitation through

Somatic tremor. This is due to tremor of the patient's body muscles, which cause a spiky irregularity of the base line, and may in places cause distortion of the cardiac complexes.

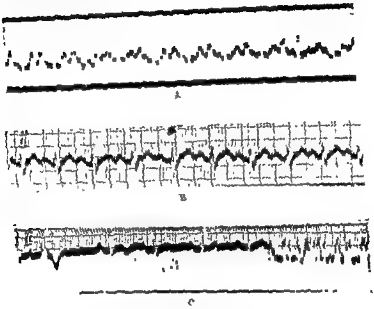


FIG 30 — Artefacts

A Somatic tremor This can be due either to tremor of the patient's voluntary muscles or to a faulty contact in which case it is analogous to the "crackling" heard in a wireless set as a result of a faulty contact

B *Electric interference* Interference from 50-cycle A C mains From a case of myocarditis with slurring of ST and abnormal sloping ST segments

C [illegible]

it is not conclusive

A similar artefact results from a defective contact ("Contact tremor", Fig 30, A)

Electric interference When cardiograms are taken in a room wired for alternating current, even though the lighting be not in use, the patient and instrument act as an aerial, picking up oscillations from the lighting circuit. This gives rise to regular sharp oscillations of the base line at a frequency corresponding to 25 or 50 cycles per second as the case may be. Often interference is slight, but sometimes it is so gross as to

any lead. Displacement of the RT interval also results from myocardial ischaemia, from pericarditis, and from drugs, particularly the digitalis group; the distinction from physiological RT displacement may be evident from the remaining cardiographic features, from the clinical features of the case, or from the fact that pathological RT displacement undergoes changes over a period of days or weeks.

Fig. 29 shows a normal series of unipolar chest leads with the corresponding unipolar limb leads and standard limb leads.

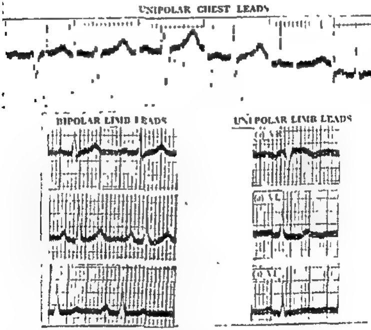


FIG. 29.—Normal cardiogram showing six unipolar chest leads (V1 to V6), three unipolar limb leads (augmented limb leads, Goldberger method), and the three standard bipolar limb leads

The direction of the deflections in the unipolar limb leads is determined by the position of the heart; in the average normal heart they are negative in VR, positive in VL and VF (see p 161).

ARTEFACTS

Before attempting to assess abnormality in the cardiogram it is essential to be acquainted with various artefacts

cleaned electrodes, necessitates excessive slackening of the fibre to give full sensitivity. In these circumstances the descending limb of R becomes slurred, merging gradually into the RT interval instead of suddenly. Slurring of R can also result from myocardial disease. A standard deflection should be recorded while the cardiogram is being exposed by introducing an E.M.F. of 1 millivolt for a short period. When slurring is an artefact the descending limb of the standard deflection is similar to that of R (Fig. 30, E), when due to myocardial disease the R waves are slurred while the descending limb of the standard deflection is sharp (Fig 30, F)

Digitalis Effects

Digitalis in therapeutic doses produces alterations in the RT segments and T waves of the cardiogram in addition to abnormalities in conduction and rhythm. Any one of its effects may occur alone, or they may be present simultaneously.

Changes in the RT segments and T waves develop successively in leads 3, 2, and 1; in a fully digitalised heart they are seen in all three leads, but are most advanced in lead 3. The earliest change is depression of RT3 which lies below the isoelectric line and is generally somewhat concave; simultaneously [illegible] ater stage T3 is inverted, [illegible] Then lead 1 is similarly [illegible] timately T is inverted in all leads Analogous depression of RT with lowering of T and ultimate inversion occurs in the chest leads. The appearances resemble those produced by ventricular hypertrophy or by myocardial ischaemia. absence of corresponding changes in QRS in the chest leads may permit differentiation; failing this the distinction depends on a knowledge of the therapeutic history of the case and conclusive evidence is obtained by noting the gradual return of the cardiogram to normal when the drug is withdrawn, the abnormality may require as long as a month to vanish entirely Examples are shown in Fig. 31, A, B, and C.

Apart from its effect on the form of the ventricular complexes, digitalis depresses the conductivity of the A–V bundle, and it may cause latent or partial block. Fig. 31, D, shows a PR interval of 0 28 second due to ouabain poisoning;

render the cardiogram illegible. It can be overcome by switching off the main supply to the house or, failing this, by earthing the leads, the instrument, and the spring mattress of the patient's bed, or the patient himself (Fig 30, B).

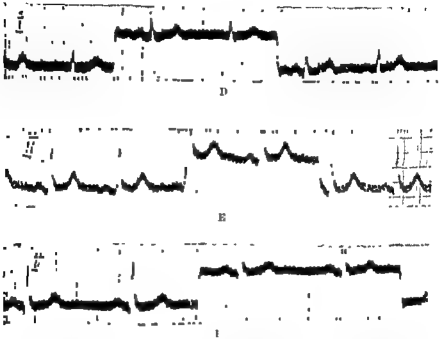


Fig 30 —Artefacts (*contd*)

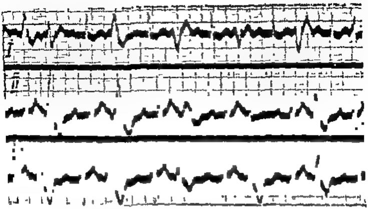
D The portable instrument was on an unsteady table The movement of the switch on throwing in the standardising L M F caused vibration and produced *undulations of the base line* resembling fibrillation waves The same result follows the termination of the standardising deflection

E [illegible]

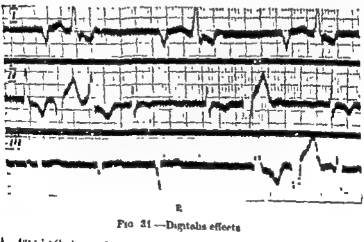
F Slurring of R due to myocardial disease In this case the descending limb of the standard deflection is sharp, while the descending limb of [illegible] is slurred Male aged 44, blood pressure 180 105, and angina of effort

Vibration In the case of portable machines, vibration of the instrument itself produces small undulations of the base line which might conceivably be mistaken for fibrillation or flutter waves (Fig. 30, D).

Slurring. High resistance in the circuit, whether due to natural resistance of the patient's skin or to inadequately



D



E

Fig 31 —Digitalis effects

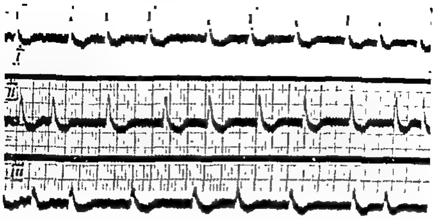
A [illegible]

B [illegible]

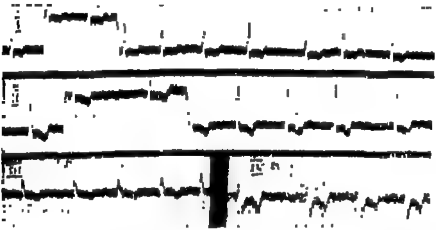
C *Auricular fibrillation* Right axial deviation [illegible]

D [illegible]

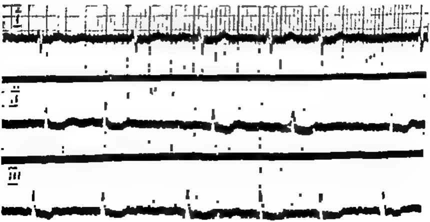
E [illegible]



A



B



C

oliguria; patients who have been stabilised for long periods on a particular dose may develop symptoms of *intoxication* if their urinary output drops. It is difficult to exaggerate the importance of keeping a close watch on persons who are under digitalis treatment.

The Electric Axis and Cardiac Vector

The heart being a three-dimensional organ, its electric axis in space ("cardiac vector") has components in three planes, viz the frontal, the horizontal, and the antero-posterior planes. The projection of the cardiac vector on the frontal plane determines the height of R and the depth of S in the limb leads, that is, the *electric axis in the frontal plane*. *The projection of the* cardiac vector on the horizontal plane determines the chest lead in which R attains its maximum height or S its maximum depth Rotation of the heart about its own axis also influences the form of QRS both the limb and chest leads.

The standard limb leads correspond to three sides of a triangle The galvanometer is so connected than an E.M.F. in the direction RA–LA, RA–LL, or LA–LL gives an upright deflection in the corresponding lead In the triangles shown (*Fig. 32*) the arrows "xy" represent an E M F., indicating its direction and magnitude The effect of this E M F. in each lead is obtained by drawing perpendiculars from the corresponding sides of the triangle to the ends of the arrow If the height of R and depth of S are measured in each lead (S being treated as a minus quantity) the following equation holds good *provided the peaks of R* (or *S*) correspond in time: (e)2 = (e)1 + (e)3 The electric axis in the frontal plane ("angle alpha") can be calculated from this equation, Einthoven's tables give the value of this *angle for all values of* (e) in the ...

... lead 2 is VF–VR, and lead 3 is VF–VL Conversely when the Goldberger technique is used, aVL is ½(lead 1 – lead 3), aVR is –½(lead 1 + lead 2) and aVF is ½(lead 2 + lead 3)

Normal axis The electric axis of the heart in the *frontal* plane corresponds roughly to the anatomical axis of the interventricular septum, it runs downwards and to the left *roughly*

ouabain is a crystalline preparation derived from strophanthus ; its pharmacological effects resemble those of digitalis

Finally, digitalis is capable of causing various forms of *arrhythmia*. Most often seen are extrasystoles These may occur at irregular intervals or an extrasystole may follow each supraventricular beat (coupled extrasystoles. pulsus bigeminus) In some cases the extrasystoles are all from a single focus and are all identical in form. A more advanced stage of digitalis poisoning is associated with the appearance of multifocal extrasystoles, differing from one another in form, while those arising from any one focus may differ in amplitude. Figs. 31, D and 31, E illustrate multifocal extrasystoles due to digitalis. If digitalis is continued beyond this point the extrasystoles occur in short runs ; paroxysms of ventricular tachycardia make their appearance , Fig. 24, B shows paroxysmal ventricular tachycardia caused by digitalis poisoning. When frequent extrasystoles or short runs of tachycardia result from digitalis overdosage the pulse is rapid and irregular , it closely resembles the irregularity of auricular fibrillation ; if the earlier stages of digitalis intoxication have been overlooked, the fatal mistake of ordering more digitalis is likely, and the result will almost certainly be death from ventricular fibrillation

In auricular fibrillation digitalis can cause coupling of the supraventricular beats, even in the absence of extrasystoles In patients with sinus rhythm and a normal or slow heart rate it occasionally produces sinus coupling, or more rarely sino-auricular block There is reason to believe that it can cause auricular fibrillation when given to a patient with simple tachycardia in the absence of cardiac failure

Clinical symptoms associated with the toxic action of digitalis are nausea, vomiting, furred tongue, dizziness, and a fall in urinary output with reappearance of oedema. The pulse is slow in many cases, and often coupled · but at times symptoms of digitalis intoxication appear when the pulse rate is still 100 or more With increasing intoxication the pulse rate rises and becomes more irregular. Patients differ greatly in their sensitivity to digitalis, some will tolerate 4 grains of digitalis leaf daily as a maintenance dose, others show symptoms of commencing intoxication after a few days on a maintenance dose of 1 grain daily. Larger doses can be tolerated during periods of diuresis than during periods of

oliguria; patients who have been stabilised for long periods on a particular dose may develop symptoms of intoxication if their urinary output drops. It is difficult to exaggerate the importance of keeping a close watch on persons who are under digitalis treatment.

The Electric Axis and Cardiac Vector

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The standard limb leads correspond to three sides of a triangle. The galvanometer is so connected than an E.M.F. in the direction RA–LA, RA–LL, or LA–LL gives an upright deflection in the corresponding lead. In the triangles shown (Fig 32) the arrows "xy" represent an E.M.F., indicating its direction and magnitude. The effect of this E.M.F. in each lead is obtained by drawing perpendiculars from the corresponding sides of the triangle to the ends of the arrow. If the height of R and depth of S are measured in each lead (S being treated as a minus quantity) the following equation holds good provided the peaks of R (or S) correspond in time: (e)2 = (e)1 + (e)3. The electric axis in the frontal plane ("angle alpha") can be calculated from this equation, Einthoven's tables give the value of this angle for all values of (e)

... lead 2 is VF–VR, and lead 3 is VF–VL. Conversely, when the Goldberger technique is used, aVL is ½(lead 1 – lead 3), aVR is –½(lead 1 + lead 2) and aVF is ½(lead 2 ...

... downwards and to the left, roughly

parallel to lead 2. R is upright in all three leads and the largest R is in lead 2 (Fig. 32 and 33, A). In the unipolar limb lead VR all deflections (P, QRS, and T) are negative, while in leads VL

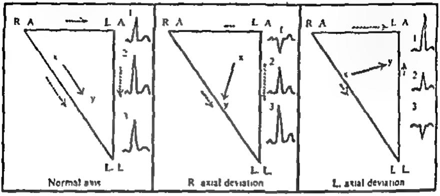
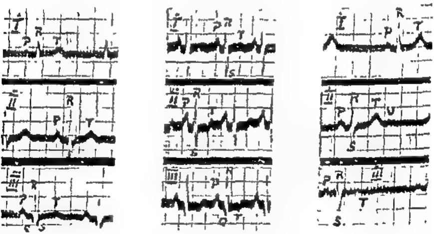


FIG 32 —Diagram illustrating the electric axis of the heart in the frontal plane



A B C

FIG 33 —The electric axis in the frontal plane.

A Normal axis B Right axial deviation C Left axial deviation

and VF they are positive (Fig 34, A) In the horizontal plane the axis runs forward and to the left, the largest R is in lead V4 (Fig. 35, B)

Right axial deviation. If the electric axis is rotated so as to run downwards, or downwards and towards the right, R becomes smaller in lead 1 and ultimately becomes inverted

(i e S becomes larger), while in lead 3, R becomes taller. The largest R is now in lead 3, the smallest R and largest S in lead 1 (Figs 32 and 33, B). The unipolar lead VR remains negative, but VL becomes negative as well; VF is more positive than before (Fig. 34, B). Right axial deviation is found in persons with a long narrow chest and a narrow vertical heart shadow in the X-ray, it is also produced by displacement of the heart to the right (e g. by left pleural effusion or pneumo-

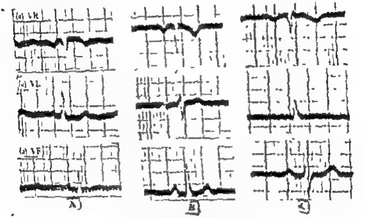


FIG 34 — Cardiograms showing the effect of the position of the heart on the unipolar limb leads

A Average type (intermediate position).
B Vertical type, or anti clockwise rotation
C Horizontal (or transverse) type, or clockwise rotation.

thorax, diaphragmatic hernia, or collapse of right lower lobe of lung, finally, it may be due to hypertrophy of the right ventricle (mitral stenosis pulmonary stenosis, emphysema, etc) in which case the additional cardiographic features described on p. 160 are present

Left axial deviation If the axis is rotated so as to run horizontally to the left, or to the left and slightly upwards, R becomes progressively smaller in lead 3 and is ultimately replaced by a deep S; while in lead 1, R becomes larger. The largest R is now in lead 1, the smallest R and largest S in lead 3 (Figs 32 and 33 C) The unipolar limb lead VF becomes negative whereas VL becomes more positive; even VR becomes less negative, sometimes positive (Fig 34, C) Left axial deviation

is found in persons with a short broad chest and flat diaphragm, in those in whom the left leaf of the diaphragm is elevated in consequence of intra-abdominal conditions (gastric tympanites, ascites, advanced pregnancy, etc.); when the heart is displaced to the left (right pleural effusion or pneumothorax,

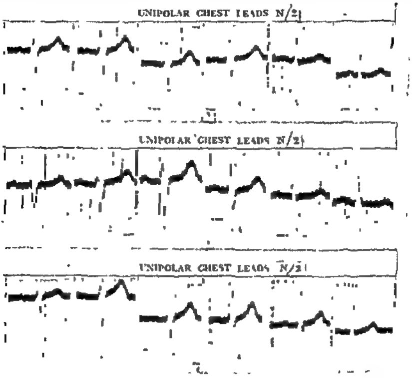


FIG. 35.—Cardiograms showing the effect of the position of the heart on the unipolar chest leads

A *Vertical heart or anti-clockwise rotation* The largest R is in lead V3
B *Average or intermediate position* The largest R is in lead V4
C *Horizontal position or clockwise rotation* The largest R is in lead V5

collapse of left lower lobe); and, finally, when there is hypertrophy of the left ventricle (aortic valvular disease, hypertension, mitral regurgitation) in which case the additional cardiographic features described in the next section (p. 166) are also present.

Rotation.—The plane of the interventricular septum determines the chest lead in which R is tallest, in the average patient this is in V4 or V5 (Fig. 35). Rotation of the heart round its long axis in an anti-clockwise direction as viewed from below (septum more nearly antero-posterior) produces tall

R waves in the right-sided chest leads V3, V2, or occasionally V1. With clockwise rotation the tallest R is in V6 or V7. Rotation can occur with or without change in the frontal plane axis

Respiratory variations in axis. When the electric axis is almost horizontal, that is, almost at right angles to lead 3, the respiratory movements of the left leaf of the diaphragm alternately raise and lower the cardiac apex. At the height of inspiration the apex is lowered, the axis runs to the left and slightly downwards and an R is present ; at the end of expiration the apex is raised, the axis runs to the left and slightly upwards and an S or Q is present in lead 3. The ventricular complexes in lead 3 show rhythmic variations with respiration, sometimes an R only, sometimes R and S, sometimes an S only. This finding in cardiograms is merely an expression of a particular degree of left axial deviation and has no pathological significance (Fig 36) In *broad-chested persons*, in *the obese*, and in others with a *transverse heart*, left axial deviation is usual and respiratory variations in axis are frequent Q3 is often enlarged but Q2 is usually absent , T3 or P3 may be inverted ; sometimes all deflections in lead 3 are inverted (Fig 37).

The Cardiac Vector and Vector Diagram—A " vector " is a line representing the direction and magnitude of a force The " cardiac vector ", which gives the direction and magnitude of the E M F. generated by cardiac activity, is a line in space ; it has projections on the frontal, the horizontal, and the sagittal (antero-posterior) planes. The arrows " x–y " in Fig 32 represent the [illegible]

[illegible] taken in these planes

Discussions and descriptions of the electric axis or of the cardiac vector are usually limited to the mean direction of the cardiac E M.F In practice, however, the direction of the cardiac potential is constantly changing throughout the cardiac cycle A whole series of successive vectors can be calculated or plotted if two leads are recorded simultaneously so that points which correspond in time can be identified in each lead A line joining the ends of successive vectors forms an irregular

is found in persons with a short broad chest and flat diaphragm; in those in whom the left leaf of the diaphragm is elevated in consequence of intra-abdominal conditions (gastric tympanites, ascites, advanced pregnancy, etc.); when the heart is displaced to the left (right pleural effusion or pneumothorax,

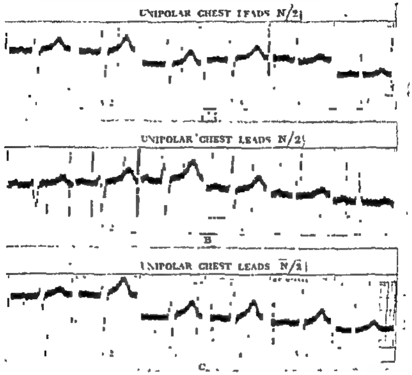


FIG 35.—Cardiograms showing the effect of the position of the heart on the unipolar chest leads

A *Vertical heart or anti-clockwise rotation* The largest R is in lead V3
B *Average or intermediate position* The largest R is in lead V4
C *Horizontal position or clockwise rotation* The largest R is in lead V5

collapse of left lower lobe), and, finally, when there is hypertrophy of the left ventricle (aortic valvular disease, hypertension, mitral regurgitation) in which case the additional cardiographic features described in the next section (p 166) are also present

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sent th…

… in Fig. 32 can be calculated f…

calc…

of t… vector on the horizontal and sagittal planes can likewise be obtained from leads taken in these planes

Discussions and descriptions of the electric axis or of the cardiac vector are usually limited to the mean direction of the cardiac E M F In practice, however, the direction of the cardiac potential is constantly changing throughout the cardiac cycle A whole series of successive vectors can be calculated or plotted if two leads are recorded simultaneously so that points which correspond in time can be identified in each lead. A line joining the ends of …

loop which is called the *vector diagram*. Normally the loop is more or less smooth, but its course may be interrupted by bays or promontories if myocardial disease causes the excitation

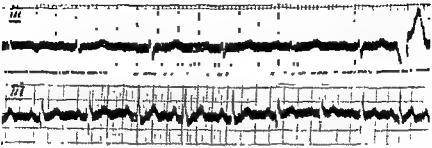
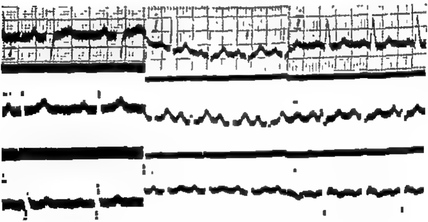


FIG. 36.—The electric axis.

Lead 3 from two different patients showing respiratory variations in axis In the upper strip the first R shows a complex of 3½ mm and no S, the third complex shows R = 2 mm, and S = 3 mm In the lower strip R becomes smaller and a deep S appears during expiration



A B C

FIG. 37.—Cardiograms in obesity and transverse heart.

A Enlargement of Q1, no Q2
B Inversion of QRS3
C Inversion of all deflections in lead 3

wave to pursue an abnormal pathway. Such a diagram may be helpful in determining whether minor notching of the QRS complexes is unimportant or significant. Minor notches are often merely an expression of the position of the heart within the chest, in which case they have no pathological significance,

but similar notching can be produced by myocardial lesions which interfere with the spread of excitation.

Calculation of the vector diagram is a time-consuming and laborious procedure, unsuitable for routine clinical use. Schellong has devised an ingenious method whereby it can be directly recorded. Three electrodes, A, B, and C, are attached to the patient's chest wall in such a position that A and B provided a bipolar horizontal lead while B and C provide a bipolar vertical lead. A cathode ray tube is fitted with two pairs of activating plates, one pair horizontal and one vertical. The horizontal electrodes A and B are connected to the horizontal plates while the vertical pair B and C are attached to the vertical plates. Potential differences in the horizontal lead displace the beam to right or left, potential differences in the vertical lead displace it upwards or downwards. At a given instant the ray is displaced to the end of the cardiac vector; and as the cardiac vector changes, the ray follows it, tracing out the vector diagram. Its movements during a single cardiac cycle are photographed on a stationary plate. An analagous diagram for the sagittal plane is recorded by pairing a vertical lead with an antero-posterior lead.

Ventricular gradient. The area enclosed by the QRS complex in a bipolar lead (A_{QRS}) is the product of the duration of QRS and its mean direction as projected on that lead. The areas enclosed by QRS in the three standard limb leads can be used to obtain a vector which expresses the duration and mean direction of the excitation wave as projected on the frontal plane ($\bar{A}_{QRS}$). Similarly the areas enclosed by ST–T in the limb leads ($\bar{A}_T$) determine a vector which expresses the duration and mean direction of the recovery (repolarisation) wave. Normally the recovery wave takes a course similar to the excitation wave but opposite in sign, so that $\bar{A}_{QRS}$ and $\bar{A}_T$ are inversely related. Thus it comes about that increase in the height and breadth of R in a particular lead (such as occurs with ventricular hypertrophy) is often associated with inversion of T in that lead.

The total areas enclosed by QRS–T in the three leads likewise determines a vector ($\bar{A}_{QRST}$) which expresses differences between the durations and courses of the excitation and recovery waves. In a healthy heart these differences are small, but with myocardial disease they may become large. This

loop which is called the *vector diagram*. Normally the loop is more or less smooth, but its course may be interrupted by bays or promontories if myocardial disease causes the excitation

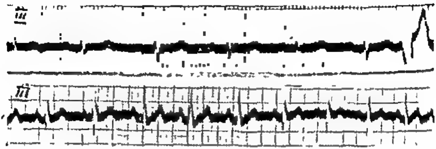
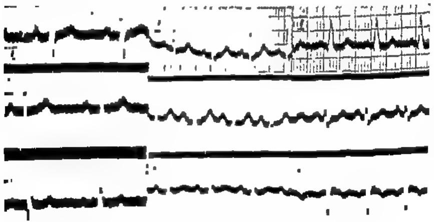


FIG 36 —The electric axis.

Lead 3 from two different patients showing respiratory variations in axis In the upper strip the first R shows a complex of 3½ mm and no S, the third complex shows R = [illegible] mm, and S = 3 mm. In the lower strip R becomes smaller and a deep Q appears during expiration



A B C

FIG 37 —Cardiograms in obesity and transverse heart.

A Enlargement of Q3, no Q2
B Inversion of QRS3
C Inversion of all deflections in lead 3

wave to pursue an abnormal pathway . Such a diagram may be helpful in determining whether minor notching of the QRS complexes is unimportant or significant. Minor notches are often merely an expression of the position of the heart within the chest, in which case they have no pathological significance ,

Finally the RT segment takes off below the iso-electric level and slopes down to end in an inverted T. These changes occur in the leads in which R becomes tall and broad, namely in leads 1 and V5 to V7 with left ventricular hypertrophy (Fig. 38)

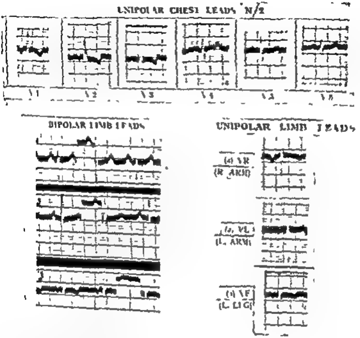


FIG 39 —Cardiogram showing right ventricular hypertrophy R is tall and T is inverted in the right sided chest leads V1 to V3 S is absent in V1 and V2 but is [illegible] axial deviation [illegible] ight type in the uni[illegible] ical of mitral steno[illegible] case and diphasic P[illegible] P1

and in leads 3 with V1 and V2 from right ventricular hypertrophy (Fig 39) Simultaneous hypertrophy of both ventricles may cause inversion of T in all three limb leads or in all seven chest leads (Fig 40)

The changes in RT and T which result from ventricular hypertrophy are sometimes referred to as indicating *ventricular strain* or *ventricular stress* (right or left, as the case may be); they were so described in the first edition of this book. The

latter vector ($\bar{A}_{QRST}$) is known as the *ventricular gradient.* It is sometimes helpful in determining the significance of minor abnormalities such as slight displacement of RT, unusually low or unusually high T waves in a particular lead, or T inversion in lead 3.

Ventricular Hypertrophy

Ventricular hypertrophy tends to cause broadening of the QRS complex from being 0·06 to 0·08 second in duration it

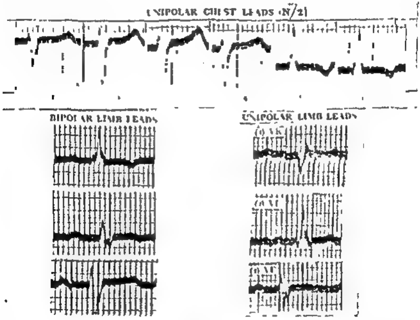


FIG 38.—Cardiogram showing left ventricular hypertrophy R tall and broad, with T inverted in leads V5 and V6 Left axial deviation with inverted T1 and T2 in standard limb leads Horizontal type in unipolar limb leads with diphasic T in (a)VF and upright T in (a)VR

may become 0·08 to 0·10 second or more If the hypertrophy affects one ventricle predominantly there is a corresponding shift in the electric axis, both in the frontal and horizontal planes. As the degree of hypertrophy increases, changes are produced in RT and T. At first the RT segment is depressed below the iso-electric line and the height of T is diminished though it still remains upright At a later stage T becomes diphasic with initial inverted and terminal upright phases.

Finally the RT segment takes off below the iso-electric level and slopes down to end in an inverted T. These changes occur in the leads in which R becomes tall and broad, namely in leads 1 and V5 to V7 with left ventricular hypertrophy (Fig 38)

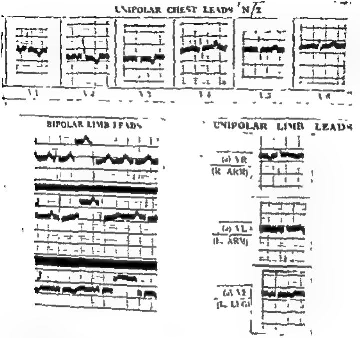
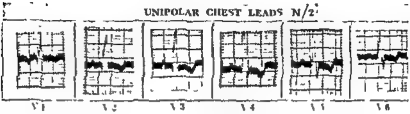


Fig 39 —Cardiogram showing right ventricular [illegible] T is inverted in the right side [illegible] and V2 but is present in the [illegible] axial deviation with inverted [illegible] limb leads Vertical type in the unipolar limb leads, with T inverted in (a)VF From a case of mitral stenosis, note the prominent P waves, slight notching of P1 and diphasic P3

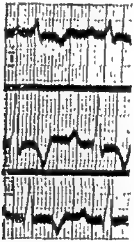
and in leads 3 with V1 and V2 from right ventricular hypertrophy (Fig 39). Simultaneous hypertrophy of both ventricles may cause inversion of T in all three limb leads or in all seven chest leads (Fig. 40).

The changes in RT and T which result from ventricular hypertrophy are sometimes referred to as indicating *ventricular strain* or *ventricular stress* (right or left, as the case may be), they were so described in the first edition of this book. The

term, however, is misleading; these changes do not necessarily indicate imminent ventricular failure, nor have they any relation to the popular concept of a "muscular strain" or a "strained



A



B

FIG 40—Cardiograms showing combined ventricular hypertrophy.

A Unipolar chest leads from a patient with atrial septal defect. R is tall and T inverted in all six chest leads

B with aves t to a

heart". As already explained (p 165), they are a direct consequence of the increased height and breadth of R with simultaneous diminution of S and Q, they are therefore merely a manifestation of ventricular hypertrophy.

BUNDLE-BRANCH BLOCK

If conduction through one branch of the A-V bundle is blocked the opposite ventricle is stimulated in the normal

manner while the ventricle on the affected side is stimulated by direct spread through the muscular interventricular septum. Conduction through muscle is slower than conduction through the Purkinje system, and QRS accordingly becomes broadened: at the same time it becomes distorted owing to the abnormal direction from which one ventricle is stimulated.

In chest leads from the affected side, the first cardiographic event is the inscription of an R due to spread of excitation

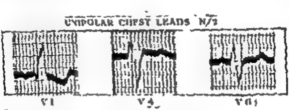


Fig. 41.—Right bundle-branch block. Chest leads V1, V4, and V6. QRS is broad (0.16 sec). R is tall and bifid in lead V1, followed by an inverted T. A broad S follows R in lead V6.

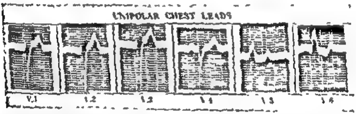


Fig. 42.—Left bundle branch block. Unipolar chest leads. QRS is broadened (0.12 sec). R is bifid in leads V5 and V6. There is a small narrow R followed by a deep broad S in leads V1 to V4. Note absence of Q in leads V5 and V6.

through the septum. The ventricular cavity now becomes negative and an S appears. Finally, spread of excitation through the outer wall of the ventricle produces a second upright wave (Second R, or R'). The result is a broad M-shaped complex; this occurs in leads V1, V2, and sometimes V3 in right bundle-branch block, or in leads V6, V5, and sometimes V4 in left bundle-branch block. The leads which show this M-shaped complex never show a Q wave since the affected ventricle does not become negative until after the spread of

excitation through the septum. Leads from the opposite ventricle (i.e. V4, V5, and V6 in right bundle-branch block, V1, V2, and V3 in left bundle-branch block) show a narrow R followed by a broad notched (or W-shaped) S (Figs. 41 and 42).

Right branch block usually produces the appearances shown in Fig. 43 in the standard limb leads. Leads 1 and 2 resemble

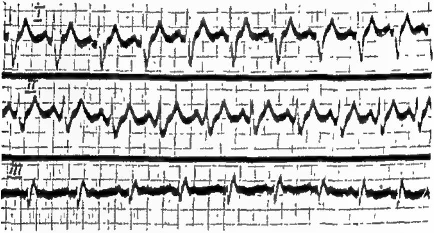


Fig. 43.—Bundle-branch block. Limb leads.

Right bundle-branch block, common type (Wilson *et alia*). S is broadened in leads 1 and 2, the total breadth of QRS amounting to 0·15 second. In lead 3, S is followed by a broad upright deflection ("second positive wave").

the chest leads from the contralateral ventricle, showing a normal R followed by a small broad S; lead 3 shows small R and S deflections followed by a second upright wave which is low and broad; the whole QRS complex measures 0·10 to 0·14 second. This pattern in the limb leads generally indicates right bundle-branch block; rarely, however, a similar pattern results from left bundle-branch block if the heart is vertical and rotated anti-clockwise. The chest leads are distinctive.

A second type of cardiogram is found in the limb leads, occasionally in right bundle-branch block and commonly with left branch block (Figs. 44 and 45). The ventricular complexes are broad and diphasic, resembling those of an extrasystole; in right branch block the initial deflection is directed downwards in lead 1 and upwards in lead 3; in left branch block the initial deflection is upright in lead 1, inverted in lead 3.

FIG 44.—Bundle-branch block Limb leads

Right bundle-branch block, rare type The ventricular complexes resemble left ventricular extrasystoles The auricles are fibrillating in this record.

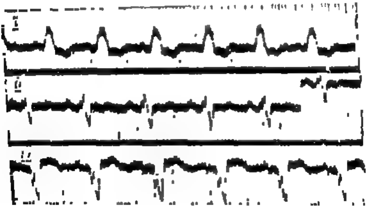


FIG 45.—Bundle-branch block Limb leads

Left bundle branch block The ventricular complexes resemble those of right ventricular extrasystoles

INTRAVENTRICULAR BLOCK (ARBORISATION BLOCK)

Sub-endocardial fibrosis or other lesions which interfere with the spread of excitation to some areas of the myocardium give rise to broadening and notching of the QRS deflections,

but falling short of the characteristic picture of complete bundle-branch block ; they are often M-shaped or W-shaped ; in the limb leads they tend to be of low voltage (Fig. 46).

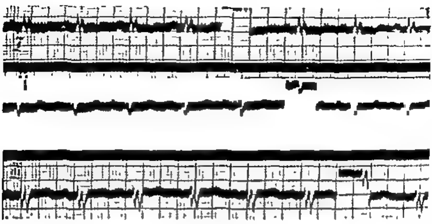


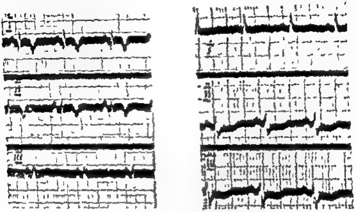
FIG. 46.—Intraventricular block. QRS is broadened in all leads. In lead 1 the complex resembles an "M", in lead 3 it resembles a "W"

Minor notching of QRS in limb leads should not be accounted pathological in the absence of definite abnormality in chest leads. Complexes in lead 3 which show a normal R and S followed by a second upright wave (second positive wave of QRS3) may be produced by a transverse heart (p 164), by bundle-branch block or by intraventricular block , chest leads will distinguish the three conditions, the first of which is not pathological.

Low-voltage Cardiograms

Low voltage in a single lead is often merely an expression of the electric axis Thus, *with a particular degree of right* axial deviation (axis approximately vertical), QRS is small in lead 1 ; with a horizontal axis, QRS is small in lead 3 , while, with a greater degree of left axial deviation, it is small in lead 2 Low voltage in a single lead may also be due to coronary occlusion as described later. The expression "low-voltage cardiogram" is not used for these cases, but only for those in which the deflections are small in all three leads, and is applied when the largest QRS is less than 5 mm. in height (Fig 47). Before attributing *pathological significance to this it is necessary*

to ensure that the instrument has been correctly standardised; a standard deflection caused by throwing in an E.M.F. of 1 millivolt should be recorded during the exposure and with the patient in circuit. Assuming standardisation to have been



A B

FIG. 47.—Low voltage cardiograms

A. *Case of coronary thrombosis* T1 and T2 are inverted (coronary T1
B. *Severe pernicious anaemia* (Hb 25% RBC 800,000) This man aged 54 was said to

correct low voltage may be due to pericardial effusion to myxoedema, or to chronic myocardial disease; the latter may be localised, but is often diffuse. The chest leads are also reduced in voltage in such cases, though not to the same extent as the limb leads Occasionally low voltage is found in a cardiogram from a normal heart, in which case it is confined to the limb leads chest leads being normal

Myocardial Ischaemia and Myocardial Infarction

Myocardial ischaemia, insufficient in degree to cause necrosis, interferes with the recovery process, producing alterations in

but falling short of the characteristic picture of complete bundle-branch block; they are often M-shaped or W-shaped, in the limb leads they tend to be of low voltage (Fig. 46).

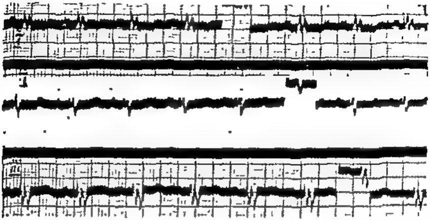


FIG 46.—Intraventricular block QRS is broadened in all leads. In lead 1 the complex resembles an "M", in lead 3 it resembles a "W".

Minor notching of QRS in limb leads should not be accounted pathological in the absence of definite abnormality in chest leads. Complexes in lead 3 which show a normal R and S followed by a second upright wave (second positive wave of QRS3) may be produced by a transverse heart (p 164), by bundle-branch block or by intraventricular block, chest leads will distinguish the three conditions, the first of which is not pathological.

Low-voltage Cardiograms

Low voltage in a single lead is often merely an expression of the electric axis. Thus, with a particular degree of right axial deviation (axis approximately vertical), QRS is small in lead 1; with a horizontal axis, QRS is small in lead 3, while, with a greater degree of left axial deviation, it is small in lead 2 Low voltage in a single lead may also be due to coronary occlusion as described later. The expression "low-voltage cardiogram" is not used for these cases, but only for those in which the deflections are small in all three leads, and is applied when the largest QRS is less than 5 mm. in height (Fig. 47). Before attributing pathological significance to this it is necessary

a deep and rather broad S, since the intrinsic deflection occurs early (Fig. 48, lead V4; R appears as a notch on the downstroke).

The changes above described occur in leads taken from a point on the chest wall overlying the infarcted area. In the

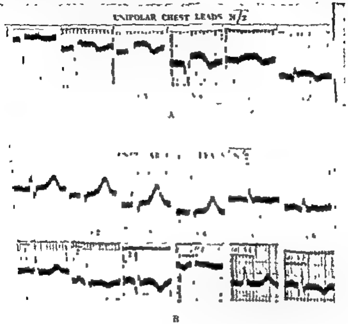


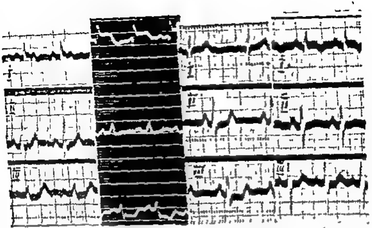
FIG 48 —Unipolar chest lead cardiograms in myocardial infarction.

majority of cases of coronary occlusion, one or more of the routine series of V leads will overlie the infarct. With posterior infarcts the changes occur in the posterior chest leads, the anterior chest leads commonly show an increase in the height

the RT segments and T waves; the spread of excitation is unaffected and the QRS complex is seldom unaltered. Severe degrees of ischaemia have the effect of elevating the RT segment above the iso-electric line: less severe grades lead to inversion of T. These changes are found in leads taken from points overlying the ischaemic area. Leads from other areas may be normal or may show depression of RT. In the limb leads RT may be depressed or elevated; if elevated in lead 1 it is commonly depressed in lead 3 and vice versa. Similarly, T may be upright or inverted, if inverted in lead 1 it is usually upright in lead 3 and vice versa. The changes are reversible, disappearing when the blood supply to the affected part becomes adequate once more. Thus they may be found during an attack of anginal pain induced by effort in a patient with narrow or rigid coronary vessels, vanishing a few minutes later when the pain has subsided in response to rest or to amyl nitrite (see Fig 71, p 341)

Myocardial infarction is associated with necrosis of the myocardium as its immediate result and with the development of a fibrous scar as its ultimate result. In either case, spread of the excitation wave through the affected area is prevented, and the QRS complex is abnormal in leads from overlying points on the chest wall. Evidence of myocardial ischaemia may or may not be present simultaneously. Leads from adjoining areas may show ischaemic changes with normal QRS complexes, or may remain entirely normal

The exact nature of the QRS complex depends on whether the infarct involves the entire thickness or merely a part of the heart wall With a complete transmural infarct neither R nor intrinsic deflection develop; there is a broad downward deflection representing Q and S fused, and followed by RT and T (Fig. 48, A, lead V3) When the infarct involves the sub-endocardial muscle while the sub-pericardial muscle remains intact, the latter is stimulated by direct spread from the neighbouring muscle, an R develops late in the QRS complex, and it is usually small owing to the reduced thickness of the surviving muscle; QRS consists of a deep and rather broad Q followed by a small narrow R (Fig. 48, A, lead V2) If the infarct involves sub-pericardial muscle while the sub-endocardial muscle remains intact the complex begins normally, Q may or may not be present; R is small and narrow, followed by



D E F G

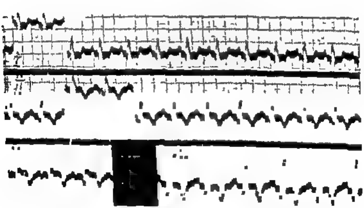
Fig 49 —Coronary occlusion, Q1,T1 type (*contd*)

D *Coronary occlusion, acute stage, Q1,T1 type* RT1 is elevated and there is a suggestion of commencing inversion of T1 RT3 is depressed In this case a single chest lead showed no abnormality

E *Coronary occlusion, acute stage, Q1,T1 type* RT1 is elevated, RT3 depressed A small Q1 is present

F

G *Coronary occlusion* Male, aged 60, who had an acute attack 24 days previously. He had a former attack at the age of 40, after which he felt quite well and was able to play golf



A

Fig 50 —Coronary occlusion, Q3,T3 type

A ...ack of coronary occlusion (un-
... Q enlarged in leads 2 and 3
...verted Lead 4R in this case
...Standardising deflections have

of T. Posterior infarcts commonly give rise to changes in lead VF which shows a prominent Q, elevated RT and inverted T. Lateral infarcts are usually reflected in lead VL as well as in one or more of the leads V4 to V7. Occasionally an infarct lies at a higher level in the myocardium, in which case special leads from points higher on the chest wall are required to demonstrate it (Fig. 69, p. 329) In the earlier stages of coronary occlusion the characteristic QRS pattern is often accompanied by RT elevation due to myocardial ischaemia; and later T inversion develops. As the infarct heals, the surviving muscle may remain ischaemic or may regain an adequate blood supply; in the former case RT displacement and/or T inversion persist, while in the latter case the RT segments and T waves revert to normal The QRS pattern is frequently permanent.

In a considerable proportion of cases of coronary occlusion though not in all, analogous changes develop in the standard bipolar limb leads Two patterns are recognised, namely the Q1,T1 type and the Q3,T3 type. In the *Q1,T1 type* (Fig 49)

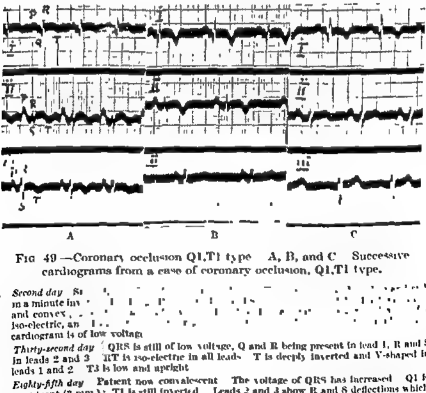


FIG 49 —Coronary occlusion Q1,T1 type A, B, and C Successive cardiograms from a case of coronary occlusion, Q1,T1 type.

A. *Second day* S[illegible] in a minute in[illegible] and convex, [illegible] iso-electric, an[illegible] cardiogram is of low voltage

B *Thirty-second day* QRS is still of low voltage, Q and R being present in lead 1, R and S in leads 2 and 3 RT is iso-electric in all leads T is deeply inverted and V-shaped in leads 1 and 2 T3 is low and upright

C. *Eighty-fifth day* Patient now convalescent The voltage of QRS has increased Q1 is prominent (3 mm); T1 is still inverted Leads 2 and 3 show R and S deflections which have become larger. T2 is now upright, and T3 has increased in amplitude

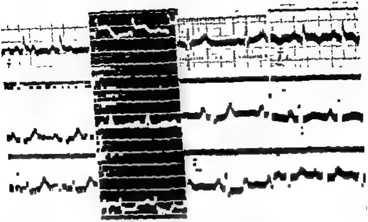
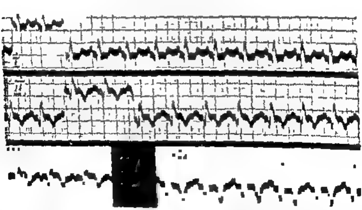


Fig. 49.—Coronary occlusion, Q1,T1 type (*contd*).

D *Coronary occlusion, acute stage, Q1 T1 type* RT1 is elevated and there is a suggestion of commencing inversion of T1 RT3 is depressed In this case a single chest lead showed no abnormality

F *Coronary occlusion, acute stage, Q1,T1 type* RT1 is elevated, RT3 depressed A small Q1 is present

E *Coronary occlusion* Male, aged 60, who had an acute attack 24 days previously. He had a former attack at the age of 40, after which he felt quite well and was able to play golf



A

Fig. 50.—Coronary occlusion, Q3,T3 type

A ... ek of coronary occlusion (un... Q enlarged in leads 2 and 3 ... inverted Lead 4R in this case ... Standardising deflections have

of T. *Posterior infarcts commonly give rise to changes in* lead VF which shows a prominent Q, elevated RT and inverted T. Lateral infarcts are usually reflected in lead VL as well as in one or more of the leads V4 to V7. Occasionally an infarct lies at a higher level in the myocardium, in which case special leads from points higher on the chest wall are required to demonstrate it (Fig. 69, p. 329). In the earlier stages of coronary occlusion the characteristic QRS pattern is often accompanied by RT elevation due to myocardial ischaemia; and later T inversion develops. As the infarct heals, the surviving muscle may remain ischaemic or may regain an adequate blood supply; in the former case RT displacement and/or T inversion persist, while in the latter case the RT segments and T waves *revert to normal* *The QRS pattern is frequently permanent.*

In a considerable proportion of cases of coronary occlusion *though not in all, analogous changes develop in the standard* bipolar limb leads. Two patterns are recognised, namely the Q1,T1 type and the Q3,T3 type. In the *Q1,T1 type* (Fig. 49)

FIG 49 —Coronary occlusion Q1,T1 type A, B, and C. Successive cardiograms from a case of coronary occlusion, Q1,T1 type.

A.

B *Thirty-second day* QRS is still of low voltage, Q and R being present in lead 1, R and S in leads 2 and 3 RT is iso-electric in all leads T is deeply inverted and V-shaped in leads 1 and 2 T3 is low and upright

C *Eighty-fifth day* Patient now convalescent The voltage of QRS has increased Q1 is prominent (3 mm), T1 is still inverted Leads 2 and 3 show R and S deflections which have become larger T2 is now upright, and T3 has increased in amplitude

As a general rule the Q1,T1 pattern in the standard limb leads is associated with anterior myocardial infarction while the Q3,T3 type occurs with posterior infarcts. Exceptions occur, however, in consequence of variations in the position or degree of rotation of the heart; and the limb leads are less

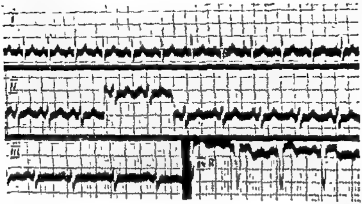


FIG 51 —Coronary occlusion with electrocardiographic changes confined to the chest lead The limb leads show slight left axial deviation, and QRS is on the borderline of "low voltage" but is otherwise normal RT is

two months previously and complained of a mild, somewhat indefinite angina of effort since.

to its

The

occasionally remain normal in coronary occlusion (Fig 51) In patients who have experienced more than one attack the limb leads may show some of the features of each pattern e g a Q3 pattern from the first attack with a T1 pattern from the second (Fig 52, p 180)

It is important to recognise that enlargement of Q and inversion of T are not confined to cases of coronary occlusion. A Q1 exceeding 2 mm in depth is pathological and is almost always due to coronary occlusion, in which circumstances R1 is small Q3 is usually enlarged in cardiograms showing right axial deviation, in the absence of right axial deviation a Q3

Q becomes prominent and R becomes small in lead 1; in the earliest stage RT is elevated in lead 1 and depressed in lead 3 Within a few hours or a few days the RT segments gradually *return to the iso-electric level* while T1 becomes inverted.

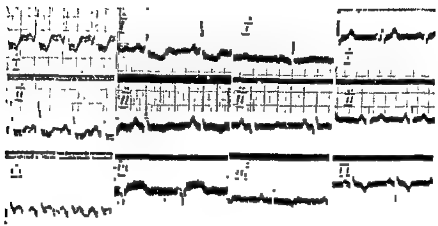


FIG 50 —Coronary occlusion, Q3,T3 type (*contd*)

B *Coronary occlusion Q3,T3 type* RT is depres[illegible]

There is left axial deviation

C [illegible]

D *Coronary occlusion, Q3 T3 type* Leads 1 and 2 show small Q waves Q3 is enlarged RT is iso-electric in all leads T2 is shallow and inverted, T3 inverted This record represents the healing or healed stage

E [illegible]

From this point the cardiogram may remain unchanged, or T1 may become upright once more after a lapse of weeks or months. The behaviour of lead 2 is inconstant. While T1 is inverted, T3 is upright and often rather pointed In the Q3,T3 type (Fig. 50) a prominent Q appears in lead 3 and usually also in lead 2; R3 is small RT is elevated in lead 3 and depressed in lead 1; later, T3 is inverted (and often T2 as well) while T1 is upright.

normal cardiograms, in many patients with "transverse heart", in right ventricular hypertrophy, in acute cor pulmonale, *in right bundle-branch block*, in some cases of pericarditis, and in cardiograms showing digitalis effects as well as in coronary occlusion of Q3,T3 type.

Pericardial Disease

In a proportion of cases of acute pericarditis changes somewhat similar to those of coronary occlusion are found. At first the RT segment is elevated in *all three standard limb leads*, or in leads 1 and 2, T remains upright. Later there may be transient inversion of T, sometimes only a terminal inversion after an initial upright phase (Fig. 53, pp. 182–3). Ultimately RT returns to the iso-electric level and T becomes upright once more. Similar changes occur in chest leads.

Pericardial effusion lowers the voltage of the cardiogram; P and T are particularly affected; they may be so flattened as to be indistinguishable, the voltage of QRS also drops. As the effusion absorbs, P and T reappear while QRS increases in voltage again.

Pericardial thickening (chronic constrictive pericarditis) causes similar lowering of the voltage of all deflections, but in this case the low voltage remains permanent unless relieved by operation (Fig. 56, p. 197).

The "Short PR–Bundle-branch-block Syndrome"

The "Short PR–Bundle-branch-block syndrome" (or "Wolf-Parkinson-White syndrome") is characterised by a short PR interval (0.10–0.12 second) with broadened and slightly notched ventricular complexes (Fig. 54, p. 184). The cardiograms are difficult to explain, since they suggest shortened A–V conduction simultaneously with delayed conduction through one of the branches of the bundle. The condition is found in persons who appear to be unusually liable to attacks of paroxysmal tachycardia, and may occur with or without signs of organic heart disease. The example shown is from a recruit referred by a National Service Medical Board who had never experienced any cardiac symptoms and in whom the

exceeding 3 mm. in depth or 30 per cent of the largest QRS deflection may be due to coronary disease, to other forms of heart disease, or to a transverse lie of the heart resulting from abdominal distension with raising of the left leaf of the diaphragm; when accompanied by a distinct Q2, coronary

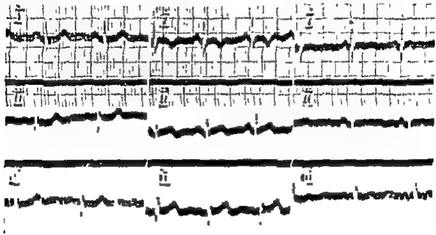


FIG. 52.—Coronary occlusion, mixed type. Serial electrocardiograms from the same patient, a male, aged 52. Two years before the acute attack he had the first of a series of relatively mild attacks of spasmodic angina which he attributed to indigestion; they gradually became more frequent and more severe, culminating in a severe attack of coronary occlusion, during and following which the series was obtained. The series of cardiograms indicate that the onset of his spasmodic angina was due to coronary occlusion with posterior infarction, the later attack being associated with anterior infarction.

A. *12 hours after onset of acute attack.* Pain still severe. Cardiogram shows deep Q waves in leads 2 and 3; RT is iso-electric and T upright in all leads. The Q waves suggest old posterior infarction.

B. *Two months later.* T has become inverted in lead 1, indicating recent anterior infarction. Q3 is still enlarged. Q2 has become smaller and R2 larger.

C. *Four months after acute attack.* Q3 remains enlarged. The inversion of T1 has disappeared and T1 is now flat. T2 and T3 are lower; it looks as though they might become inverted at a later date. The enlarged Q3 is the only positive evidence of coronary occlusion now remaining.

occlusion is *the most likely cause (85 per cent of cases).* When lead 3 shows a downward deflection only, it should be labelled Q if lead 2 shows Q and R deflections, or S if lead 2 has R and S deflections. The initial limb of a W-complex in lead 3 may be regarded as Q. T1 is inverted, not only in coronary occlusion, but also in left ventricular hypertrophy, in left bundle-branch block, in some cases of pericarditis, and in cardiograms showing advanced digitalis effects. T3 is inverted in some

normal cardiograms, in many patients with "transverse heart", in right ventricular hypertrophy, in acute cor pulmonale, in right bundle-branch block, in some cases of pericarditis, and in cardiograms showing digitalis effects as well as in coronary occlusion of Q3,T3 type.

Pericardial Disease

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Pericardial effusion lowers the voltage of the cardiogram; P and T are particularly affected, they may be so flattened as to be indistinguishable, the voltage of QRS also drops. As the effusion absorbs, P and T reappear while QRS increases in voltage again

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Dextrocardia.—All deflections inverted in lead 1. Leads 2 and 3 transposed (Fig. 89, p. 430). The cardiogram can be "faked" by transposing the right and left arm leads in a normal individual

Other congenital lesions, if they produce any change at all, usually give rise to right ventricular hypertrophy (Fig. 86, p. 421). A notable exception is the case of tricuspid atresia with rudimentary right ventricle in which left axial deviation is the rule. Coarctation of the aorta usually, and patent ductus arteriosus sometimes causes evidence of left ventricular hypertrophy.

It will be recognised that the electrocardiograph gives valuable information in many cases, and that, in difficult or doubtful cases, it may clinch the diagnosis The instrument, however, is in no wise a "penny in the slot machine"; it cannot replace clinical examination, and, in the absence of the latter, it may on occasion prove misleading.

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SECTION 2. ANATOMICAL LESIONS

CHAPTER 7

PERICARDITIS

AETIOLOGY.—*Acute rheumatism* is by far the most common cause. The primary lesion is probably a myocarditis, from which pericarditis arises by direct extension In many cases there is endocarditis as well. Alternatively, pericarditis may occur with a later attack of acute rheumatism in a person whose valves have been damaged in an earlier attack. Tonsillitis is sometimes followed by pericarditis without the occurrence of joint pains; these cases are probably rheumatic.

Less frequently, pericarditis is caused by other acute specific fevers, including scarlet fever, pneumonia, influenza, typhoid fever, diphtheria, and smallpox. Septicaemia or pyaemia may also be complicated by pericarditis

Tuberculous pericarditis usually arises by extension from tuberculous mediastinal glands, it may precede, accompany, or follow pulmonary or pleural infection, it is therefore sometimes found in the absence of any demonstrable lung or pleural lesion. Occasionally it arises as a blood infection, an incident in the course of miliary tuberculosis.

Septic pericarditis is the result of direct extension of infection from neighbouring structures. It may follow infected wounds of the chest wall, empyema, septic peritonitis, or rupture into the pericardium of a bronchiectatic cavity, an oesophageal carcinoma, or a septic gland

In some cases no primary cause is found, and the pericarditis appears to be *primary* or *idiopathic*. Some would regard these cases as rheumatic, some as tuberculous. An apparently primary pericarditis is sometimes associated with pleurisy, peritonitis, or both; these cases again may be rheumatic, tuberculous, or of unknown aetiology; they are described as *Polyserositis* or " Concato's Disease ".

Localised pericarditis occurs as a sequel to coronary thrombosis when the infarcted area of myocardium reaches the pericardial surface. In these circumstances the pericarditis is

sterile. Occasionally a coronary thrombosis is followed by a generalised pericarditis

Malignant pericarditis is due to invasion of the pericardium by malignant growths in the neighbourhood, or to blood-borne metastases. In these cases again the pericarditis is sterile.

Pericarditis is most common in children since acute rheumatism is the most frequent cause; but it may occur at any age, and in either sex. It is not uncommon as a terminal infection in chronic nephritis, gout, diabetes, and malignant disease. The organisms concerned in causing pericarditis are various and numerous; they include streptococci, staphylococci, pneumococci, typhoid bacilli, tubercle bacilli, etc. In some cases the pericarditis is sterile

CLASSIFICATION.—Pericarditis may be acute or chronic. Acute cases are classified as "dry" (fibrinous), or "with effusion"; the effusion may be serous, haemorrhagic, or purulent, the latter being described as a "pyopericardium". Chronic pericarditis occurs in two forms known respectively as "adhesive" and "constrictive".

PATHOLOGY

Acute Pericarditis.—The earliest change is loss of lustre and dryness of the visceral layer of the pericardium, with hyperaemia This may be followed by deposition of a fibrinous exudate (dry pericarditis) which forms a shaggy layer over the surface of the heart ("Bread and Butter Pericardium"); or the fibrinous exudate may be scanty and in its place there is a fluid exudate, in most cases serous but sometimes haemorrhagic or purulent Rheumatic cases are usually associated with a fibrinous or a serous exudate; often there is a fibrinous exudate early with serous effusion later Haemorrhagic exudate is most frequent with malignant pericarditis, but occasionally occurs in tuberculous cases and sometimes in influenzal cases. Purulent exudate is most likely in septic pericarditis due to pyogenic cocci, in septicaemia, in pyaemia, or in pneumonia

The pericarditis in rheumatic cases is accompanied [illegible] diffuse [illegible] valvu[illegible] to the [illegible] underlying the pericardium.

Death during the acute stage may result from the general

toxaemia in septic or tuberculous cases, from the associated myocarditis in rheumatic cases, or from "cardiac tamponade" in cases with effusion irrespective of the aetiology. If the patient survives the acute stage, the exudate is absorbed in many cases (whether fibrinous or fluid). Resolution can be complete and it is often impossible to detect any permanent change in the pericardium; or there may be localised patches of thickening ("milk spots") which do little damage. On the other hand the pericarditis sometimes becomes chronic.

Chronic pericarditis is in some cases a sequel to an acute attack, other cases are chronic from the outset. There are *two types with totally different effects on the heart, known as* adhesive and constrictive.

Chronic Adhesive Pericarditis.—Adhesions form, on the one hand, between the two layers of the pericardium, and on the other hand between the parietal layer and the adjacent structures including the chest wall. A few slender adhesions connecting parietal and visceral layers do little damage and do not seriously interfere with the efficiency of the heart. When, however, there are numerous dense adhesions (in some cases the two layers are so densely matted together that their separation is quite impossible), and when in addition the parietal layer is adherent to the chest wall in front, the lungs laterally, and the posterior mediastinal structures and spine behind, there is serious interference with cardiac systole. The heart becomes enormously hypertrophied in an attempt to overcome this difficulty—some of the largest hearts on record are the result of adhesive pericarditis. With each systole or attempt thereat, considerable traction occurs on the fibrous auriculo-ventricular ring as well as on the chest wall. The heart becomes dilated; I have seen a mitral stenosis with a "buttonhole" mitral valve dilated until it would admit three fingers by this process, and the chest wall not infrequently becomes deformed. The cardiac reserve is grossly impaired, and cardiac failure is usually progressive. In some cases the adhesions cause gross portal obstruction producing the clinical picture known as "pericarditic pseudo-cirrhosis of the liver" or "Pick's disease". In other cases adhesions may interfere with the coronary circulation.

Chronic Constrictive Pericarditis.—In this type there are no adhesions between the two layers of the pericardium, but the parietal layer becomes much thickened and fibrous, it

may reach half an inch in thickness and it may be as tough as leather. The visceral layer may also be thickened, though usually to a lesser degree. In some cases deposits of calcium salts are formed in it ("calcification of the pericardium"). Many of these cases are tuberculous in origin; others follow a septic infection; in some the aetiology remains obscure. Myocardial lesions are usually absent, though occasionally present in tuberculous cases, endocardial lesions are invariably absent, and the available evidence suggests that chronic constrictive pericarditis is never rheumatic in origin. This type of chronic pericarditis interferes with diastole and not with systole; it *prevents adequate filling of the heart in diastole and causes* venous congestion. There is no enlargement of the heart; on the contrary, the heart is often relatively small. Like adhesive pericarditis, the constrictive type often causes gross portal congestion simulating cirrhosis of the liver (Pick's disease).

SYMPTOMS, PHYSICAL SIGNS, AND TREATMENT

Acute Dry Pericarditis.—There are often symptoms of the cause, for example, acute rheumatism. There may be *no other symptoms, or merely fever, the pericarditis being found only on routine examination of the heart* In some cases *the development of pericarditis is characterised by pericardial pain.* Occasionally the associated myocarditis leads to sudden development of congestive failure; to sudden severe dyspnoea, or to an attack of acute pulmonary oedema. Occasionally there are true anginal attacks.

The *physical sign* is friction, which may be palpable as well as audible It may be heard over any point of the praecordium, but most often appears first at the base of the heart and lasts longest there It is often transient, lasting only a few hours and disappearing when an effusion develops, and it is easily missed unless the heart is repeatedly examined Other physical signs, if present, are not due to the pericarditis but to the associated myocarditis, or to accompanying endocarditis The electrocardiogram shows elevation of the RT interval in two leads or in all three leads in about half the cases (Fig 53, pp 182–3)

The *treatment is rest in bed, nursing,* and relief of pain if present. Relief is often given by counter-irritation, especially

a mustard leaf, a blister, or leeches; sometimes an ice-bag helps. If these measures fail to give relief, morphine should be given without hesitation to an adult, or nepenthe to a child; and the dose should be sufficient to relieve the pain. The general nursing measures, diet, period of rest, and treatment during convalescence are the same as in rheumatic endocarditis and are described on pp. 242–3.

Pericarditis with Effusion.—The illness starts as a dry pericarditis, the effusion developing within a few hours or after a few days Pain, if present, is replaced by a dull ache or sense of weight, while friction disappears or remains audible only at the base. Fever persists, and may be accompanied by restlessness, insomnia, or delirium. The blood usually shows a leucocytosis. The occurrence of rigors points to a purulent effusion as does also a high leucocytosis. Small effusions cause few local symptoms and often remain unsuspected. Large effusions cause pressure on surrounding structures giving rise to respiratory distress, pressure cough, dysphagia, or obstruction of the superior vena cava leading to oedema of the neck, face, or arms, in some patients the appearance suggests a mediastinal tumour, while in others the resemblance to nephritis is very close.

Symptoms of cardiac failure due to *tamponade* depend on the rapidity with which the fluid accumulates rather than on its amount, as little as 200 to 300 c c may cause death if very rapidly developed (the most striking examples being cases of haemopericardium), whereas effusions of 2 litres have been recorded without failure in cases where the accumulation of fluid was slow Increased intrapericardial pressure affects the thin-walled veins to the greatest extent, the auricles next, and the ventricles least. The inferior vena cava and hepatic vein are especially liable to be obstructed owing to their anatomical position, the result is systemic and portal venous congestion with a fall in cardiac output The pulmonary veins may also be compressed, there may or may not be pulmonary congestion, depending on the sequence in which the veins are affected. The patient develops oedema, which may include the face as well as the dependent parts, distension of the neck veins, and enlargement of the liver; pulsation in the distended neck veins and engorged liver is slight or absent, in contrast with other forms of congestive failure. When pulmonary congestion is present there is severe dyspnoea or orthopnoea in addition to

the foregoing symptoms The pulse is small and rapid with low systolic and pulse pressures. Pallor is an important sign indicating necessity for paracentesis; there may be a mixture of pallor and cyanosis, less often a livid cyanosis When pericarditis complicates valvular disease, tamponade leads to intensification of any pre-existing congestive failure.

Physical signs The apex impulse is usually invisible and often impalpable, otherwise it is diffuse, feeble, and displaced outwards and upwards. Occasionally there is some fullness in the precordial area The distended neck veins tend to collapse during inspiration A characteristic change in cardiac dullness can be demonstrated; the upper border is displaced upwards reaching the second space, second rib or even higher; the left border sweeps downwards and outwards in a convex line to the apex, the right border runs downwards and outwards to form an obtuse angle with the liver dullness (Rotch's sign, i e obliteration of the cardio-hepatic angle) The heart sounds are muffled and distant, while friction has usually disappeared or remains audible only at the base; murmurs may be present, but are due to associated endocarditis or valvular lesions, not to the pericardial effusion. In addition there are signs of atelectasis affecting the lower lobe of the left lung, or occasionally both lungs, these appear first at the angle of the scapula, and as the effusion increases, they spread over a greater area of the lower lobe There is impaired resonance with tubular respiratory murmur and slightly increased vocal fremitus—signs which may be mistaken for consolidation if the pericardial effusion has not been recognised In most, though not all, cases with a large effusion, the pulse volume diminishes during inspiration and increases during expiration, in extreme cases the pulse may disappear entirely towards the end of a deep inspiration This is the reverse of what occurs in health (see p 100), and is described as a "pulsus paradoxus"

The *diagnosis* from hypertrophy or dilatation of the heart is suggested by the shape of the cardiac dullness, the obliterated impulse, and the muffled sounds. The signs at the base of the left lung may lead to a mistaken diagnosis of consolidation. The electrocardiogram with pericardial effusion usually shows a drop in voltage, especially of the P waves and T waves; the latter are often invisible; sometimes they still show slight elevation of the RT interval, terminal inversion of T, or simply

a mustard leaf, a blister, or leeches; sometimes an ice-bag helps. If these measures fail to give relief, morphine should be given without hesitation to an adult, or nepenthe to a child; and the dose should be sufficient to relieve the pain. The general nursing measures, diet, period of rest, and treatment during convalescence are the same as in rheumatic endocarditis and are described on pp. 242–3.

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Symptoms of cardiac failure due to *tamponade* depend on the rapidity with which the fluid accumulates rather than on its amount: as little as 200 to 300 c.c. may cause death if very rapidly developed (the most striking examples being cases of haemopericardium); whereas effusions of 2 litres have been recorded without failure in cases where the accumulation of fluid was slow. Increased intrapericardial pressure affects the thin-walled veins to the greatest extent, the auricles next, and the ventricles least. The inferior vena cava and hepatic vein are especially liable to be obstructed owing to their anatomical position, the result is systemic and portal venous congestion with a fall in cardiac output. The pulmonary veins may also be compressed; there may or may not be pulmonary congestion, depending on the sequence in which the veins are affected. The patient develops oedema, which may include the face as well as the dependent parts, distension of the neck veins, and enlargement of the liver, pulsation in the distended neck veins and engorged liver is slight or absent, in contrast with other forms of congestive failure. When pulmonary congestion is present there is severe dyspnoea or orthopnoea in addition to

infecting the latter is avoided; in this situation the needle should be directed backwards, upwards, and slightly outwards. The danger of perforating the pleura is less when paracentesis is performed close to the sternal border than at the apex; in either of these situations the needle is inserted at right angles to the chest wall. Whichever situation is chosen, local anaesthesia with novocain is advisable.

Purulent pericarditis (pyopericardium) is best treated by open surgical drainage coupled with sulphonamides or penicillin.

Some physicians recommend the giving of diuretics to promote the absorption of a serous effusion, but it is doubtful whether they have any effect whatever in this direction.

Chronic Adhesive Pericarditis.—There is usually a history of several attacks of acute rheumatism, one or more of which has been complicated by pericarditis. Progressive deterioration in the cardiac reserve develops, sometimes immediately after the acute attack, sometimes after a considerable interval. Occasionally there are attacks of anginal pain from obstruction of the coronary vessels by adhesions. Some cases complain of swelling of the abdomen due to portal obstruction.

Physical signs. Distended veins are seen in the neck, and they collapse on inspiration. There is frequently deformity of the precordial ribs, and sometimes of a considerable part of the left side of the chest; systolic retraction is visible in the precordial area or at the apex, occasionally over the lower ribs posteriorly. The apex is displaced outwards, often in the axillary line; it remains fixed when the patient turns on his side. Percussion simply shows considerable enlargement of the cardiac dullness. The findings on auscultation are not characteristic; there may be regurgitant murmurs at various valves due to dilatation, or stenotic murmurs due to coincident valvular lesions. In many cases there is a typical "pulsus paradoxus." It should be emphasised that these signs are not all present in every case—there may be only one or two of them, and the diagnosis is not always easy; but if they are all present or even the majority, diagnosis is simple. Electrocardiograms usually show high voltage, but in other respects they are not characteristic.

Treatment. Cardiac failure must be treated by rest, simple impairment of reserve by restriction of activity. Digitalis may be of some help along with rest when congestive failure is

a shallow inverted T wave. X-ray shows pear-shaped or conical enlargement of the heart shadow (Fig. 15, p. 92), and, on screening, the pulsations are of small amplitude. When constitutional symptoms are mild or absent, tuberculosis should be suspected.

The *treatment* of pericarditis with effusion includes rest in bed, nursing, and treatment of the cause. In many cases the cause is acute rheumatism, and the general treatment is that described on pp. 241–4. Where the condition is due to pneumococci or streptococci, and in septic cases secondary to infected wounds or to septicaemia, sulphonamides or penicillin should be given. Of the sulphonamides, sulphamezathine or sulphadiazine seem to be less liable to cause nausea and vomiting than other members of the series; the initial dose of either of these drugs should be 3 grams (6 tablets) followed by 2 grams every six hours Penicillin may be given in doses 500,000 to 1,000,000 units daily in three or four divided doses; when doses of this magnitude are employed, the necessity for an intravenous drip or for frequent (three-hourly) intramuscular injections is obviated. In all cases, symptomatic treatment may be required for insomnia or for pain should this persist; chloral hydrate is probably the most effective and safest sedative for insomnia or restlessness which are not associated with pain, pain is treated as in dry pericarditis. Paracentesis is indicated as a diagnostic procedure if a purulent effusion is suspected owing to the occurrence of rigors or a high leucocytosis: a purulent effusion should be drained surgically. A therapeutic paracentesis should be performed in the following circumstances :—(1) If the effusion is large and is causing distress in breathing or pressure symptoms; (2) if it is causing progressive cardiac failure, (3) if the effusion is increasing rapidly so that it is likely to cause distress or failure unless relieved, and (4) if, despite adequate rest and general treatment, the effusion shows no signs of absorption after 14 days In performing paracentesis the needle may be inserted either in the angle between the xiphoid cartilage and the left costal margin, in the 5th interspace close to the left border of the sternum, or in the 5th interspace just medial to the apex and the left border of cardiac dullness. Paracentesis through the substernal angle is stated to be the most safe, particularly where a purulent effusion is suspected, as the needle does not traverse the pleura and the risk of

case Unfortunately the operation is a difficult one and carries a high mortality. Good results have been obtained by surgeons experienced in its use; but as the condition is rather rare, comparatively few surgeons have had the opportunity of acquiring the necessary experience A period of preliminary

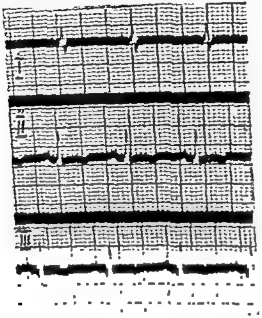

FIG 56—From a case of tuberculous pericarditis; the patient, a male, aged 23, was admitted with fever and pericardial friction, later he developed a serous effusion which slowly absorbed, leaving a thickened pericardium with symptoms and signs of constrictive pericarditis (verified at operation)

Four months after onset. Effusion now completely absorbed but signs of constrictive pericarditis present P waves low T1 flat T2 and T3 inverted The voltage of QRS is reduced to 50 per cent of its value in the early stages of the illness

rest and observation is necessary to ensure that the disease is quiescent; the criteria are the same as in the case of adhesive pericarditis Operation consists of resection of part of the thickened pericardium, sufficient must be removed to allow the heart to expand Tudor Edwards stresses the necessity of incising the visceral layer as well as the parietal layer. If operation is impracticable owing to the patient's general state of health, treatment can only be palliative and the results are disappointing

present, but is often disappointing as the difficulty is so largely mechanical. Operative treatment should be considered; it involves removal of sufficient portions of ribs to free the heart from its attachment to the chest wall; the attachment to the lungs is of less importance. Before operating it is essential to make certain that the disease is quiescent by a period of observation; temperature and blood sedimentation rate must be normal, the patient's general health must be good, and any anaemia must be corrected. As many cases are slowly progressive with constant low-grade activity, few turn out to be suitable for operation.

Chronic Constrictive Pericarditis.—Tuberculosis is the most frequent cause The disease is often chronic from the outset; rarely it follows an acute attack. Symptoms develop insidiously, enlargement of the liver with progressive distension of the abdomen from ascites are often the first features to attract the patient's attention. Not infrequently these symptoms remain out of proportion to other signs of cardiac failure throughout the entire illness (Pick's disease) In other cases oedema of the ankles and cyanosis accompany the abdominal distension. the oedema gradually extends to the level of the lower thorax where it may stop abruptly. In contrast with other forms of cardiac failure, breathlessness is usually slight and sometimes absent

Physical signs. The apex impulse is invisible and impalpable The neck veins are distended but not pulsating On percussion, the cardiac dullness is normal in extent or only very slightly enlarged. On auscultation, the heart sounds are very faint, sometimes quite inaudible At X-ray examination, on screening, pulsations are scarcely visible along the borders of the heart, the thickened pericardium occasionally causes backward displacement of the oesophagus simulating mitral stenosis Cardiograms are of low voltage, they may show any of the features described under pericardial effusion (p. 193, and see Fig. 56).

Diagnosis. Congestive failure and portal obstruction with cardiac dullness of normal size, feeble or inaudible heart sounds, [illegible] electrocardiogram [illegible] signs in a case of brown atrophy of the heart.

Treatment. Operation offers the only hope of cure in this

case Unfortunately the operation is a difficult one and carries a high mortality. Good results have been obtained by surgeons experienced in its use; but as the condition is rather rare, comparatively few surgeons have had the opportunity of acquiring the necessary experience. A period of preliminary

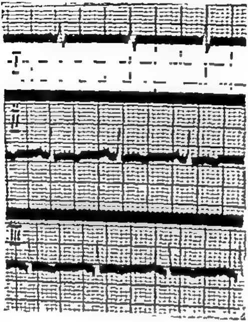

Fig 58. F...

Four months after onset Effusion now completely absorbed but signs of constrictive pericarditis present P waves low T1 flat T2 and T3 inverted The voltage of QRS is reduced to 60 per cent of its value in the early stages of the illness

rest and observation is necessary to ensure that the disease is quiescent, the criteria are the same as in the case of adhesive pericarditis Operation consists of resection of part of the thickened pericardium, sufficient must be removed to allow the heart to expand Tudor Edwards stresses the necessity of incising the visceral layer as well as the parietal layer If operation is impracticable owing to the patient's general state of health, treatment can only be palliative and the results are disappointing

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CHAPTER 8

ENDOCARDITIS

AETIOLOGY.—(1) *Acute rheumatism* is much the most common cause, endocarditis is present in probably 75 per cent of the cases of acute rheumatism in children, but in adults the heart has a considerably better chance of escaping. Endocarditis similar to that caused by acute rheumatism may occur after *tonsillitis* even in the absence of joint pains, in children suffering from *subacute rheumatism*; or occasionally without definite evidence of its rheumatic nature—primary or idiopathic endocarditis. Acute endocarditis is relatively rare with *acute chorea*, but many patients who have an attack of chorea also have an attack of endocarditis either beforehand or at a later date, and many others subsequently develop a chronic endocarditis even though there has been no evidence of endocarditis during the acute attack of chorea.

(2) *Scarlet fever.* In some cases scarlet fever is followed by joint pains, probably identical with acute rheumatism, and in these cases there may be a simple endocarditis. In rare cases scarlet fever is followed by an endocarditis which corresponds in type to a subacute bacterial endocarditis.

(3) *Other acute infections* may cause endocarditis, especially streptococcal infections; it also occurs in specific infections such as pneumonia, gonorrhoea, cerebrospinal fever, typhoid, and in streptococcal or staphylococcal septicaemia. *In these* cases the endocarditis usually conforms to the malignant type

Endocarditis is especially prone to affect valves which have already been damaged by a former attack Thus when a valve is affected and damaged in one attack of acute rheumatism, further damage usually occurs with each subsequent attack. Valves which have been damaged by rheumatic endocarditis are more prone to a superimposed subacute or acute bacterial endocarditis Congenital lesions also increase the susceptibility to endocarditis; a subacute bacterial endocarditis may become engrafted on a congenital valvular lesion, a congenital septal defect, or a congenital patent ductus arteriosus. Never-

theless a healthy heart may be affected, indeed often is affected in a child with an initial attack of acute rheumatism.

CLASSIFICATION.—Endocarditis is classified as follows:

Acute Endocarditis.—

Simple endocarditis corresponding roughly to clinical rheumatic endocarditis.

Subacute bacterial endocarditis corresponding roughly to clinical endocarditis lenta.

Acute ulcerative endocarditis corresponding roughly to clinical malignant endocarditis.

Chronic Endocarditis.—A slowly progressive form, causing slowly progressive damage to the valve cusps.

Chronic Stationary Valvular Lesions.—Scarring and deformity which have resulted from endocarditis which has healed; in the absence of fresh infection, the lesions do not become worse.

ACUTE SIMPLE OR RHEUMATIC ENDOCARDITIS

PATHOLOGY.—Small deposits of fibrin with entangled leucocytes form on the surface of the valve cusps at the "line of contact", i.e close to the free margin on the auricular surface of the mitral cusps or the ventricular surface of the aortic cusps; they are known as "warty vegetations". There is an associated diffuse myocarditis with "Aschoff nodules" (collections of endothelial cells, some of which are multinucleate, lymphocytes, polymorphs, and often eosinophil cells); these are found especially in the subendocardial zone and along the course of the blood vessels in the myocardium There may be a pericarditis as well, but not always

The vegetations are firmly attached to the valve cusps, and are not easily broken off, therefore emboli are rare unless auricular fibrillation has been present and has led to thrombosis in the auricular appendages. Blood cultures are usually sterile, and so are the vegetations.

Later, vascularisation develops at the base of the valve cusp; new vessels grow in from the myocardium. The condition tends to heal In some cases healing occurs with relatively little damage to the valve cusp, the vegetations are absorbed and there remains merely slight thickening of the

free margins of the cusp; this may be so slight as to be scarcely recognisable in later years, or it may be easily recognisable though insufficient to interfere with the normal function of the cusp. In other cases, healing is associated with a much greater degree of thickening and deformity of the valve cusps which may be incapable of closing the valve orifice causing incompetence or regurgitation. In many, the cusps become adherent to one another as well as thickened, resulting in stenosis of the valve orifice

Repeated attacks of acute rheumatism are frequent. The first attack sometimes leaves only minimal damage to the valves but this may be increased with each subsequent attack till severe damage has resulted Alternatively, in some cases after subsidence of the acute attack, a chronic endocarditis persists, which leads slowly (over a period of years) to progressive thickening and deformity of the cusps.

The valves are affected in the following order of frequency (1) Mitral valve. (2) Mitral and aortic. (3) Aortic alone (4) Occasionally mitral, aortic, and tricuspid. (5) More rarely, mitral aortic and pulmonic. It is very rare for all four valves to be affected, and quite exceptional for the valves on the right side to be affected without any involvement of the valves on the left.

Chronic endocarditis, in the case of the mitral valve, tends to cause fusion and thickening of the cusps, coupled with thickening and shortening of the chordae tendineae. The fused cusps form a diaphragm across the mitral orifice, with a small aperture in the centre ("Button-hole mitral stenosis"), the aperture is often so small as to admit only the tip of one finger In some cases the shortened chordae draw the diaphragm formed by fusion of the cusps into a funnel shape ("Funnel-shaped mitral stenosis"), again, the orifice at the end of the funnel may admit only one finger or its tip In consequence of the obstruction the left auricle becomes dilated and hypertrophied; this rarely suffices to compensate the lesion, and the pulmonary circulation becomes engorged, with raised pressure in the pulmonary artery; the right ventricle becomes hypertrophied and compensates the lesion. Note that the left ventricle does not undergo hypertrophy unless the mitral valve is incompetent as well as stenosed, or unless the aortic valve is affected as well

Thickening and distortion of the aortic cusps lead to regurgitation frequently, to stenosis and regurgitation occasionally. The lesions are compensated by hypertrophy of the left ventricle, in the case of stenosis a concentric hypertrophy, in the case of regurgitation (or stenosis with regurgitation) a combination of dilatation and hypertrophy giving rise to a cup-shaped thickened ventricle.

Similar considerations apply in the rarer cases of right-sided valve involvement.

Patients dying in the acute stage of endocarditis usually show little or no evidence of congestive failure, but may instead show evidence of an acute infection, or evidence of acute rheumatism. Patients dying with chronic valvular lesions usually show *signs of chronic congestive failure in addition to* the findings in the heart itself. In many cases of chronic endocarditis the final stage results either from a superimposed bacterial endocarditis or from auricular fibrillation, each of which causes its own characteristic pathological findings

Symptoms.—In most cases acute endocarditis accompanies the attack of acute rheumatic fever, and the symptoms are completely overshadowed by those of the primary disease—*fever, joint pains, and swelling which affect the larger joints* and move about from place to place, and in some cases subcutaneous nodules. In these circumstances the endocarditis is recognised only in consequence of repeated examinations of the heart. When endocarditis follows tonsillitis, or when it develops in the absence of tonsillitis or acute rheumatism, the symptoms are those of an infection—general malaise and weakness, loss of appetite, disinclination for effort, fever especially in the evenings, and sweats The leucocyte count may be normal or slightly raised. Pain is not a symptom, unless there is pericarditis. In some cases there is abdominal pain at the onset, and this may lead to a mistaken diagnosis of appendicitis or other acute abdominal lesion Breathlessness and congestive failure are uncommon in the acute stage, occasionally they develop when acute endocarditis occurs in a patient whose heart has previously been damaged by former attacks, in progressive cases, or when endocarditis is accompanied by pericarditis with effusion

Physical Signs.—*First stage* The impulse is diffuse and extends beyond *the nipple line*, *there is enlargement of* cardiac

dullness to the left; the first sound is soft; a soft systolic murmur accompanies the first sound at the apex; the second pulmonic sound is accentuated. These physical signs are probably due to the associated myocarditis and do not of themselves indicate endocardial involvement. When all are present, the diagnosis of carditis is certain; but one or more may be missing, and sometimes only one or two are present, in which case diagnosis is difficult. The cardiogram may be normal or may show slight lengthening of the PR interval to beyond 0.20 second, and the T waves are often somewhat lowered. With convalescence these signs may disappear completely.

Second stage In addition to the foregoing signs others appear Mitral incompetence is accompanied by an alteration in the systolic murmur which becomes louder, harsher, and more highly pitched. Mitral endocarditis or commencing mitral stenosis is shown by the appearance of a diastolic murmur, this is at first short and may simulate reduplication of the second sound at the apex; later it becomes an undoubted mid diastolic murmur. Aortic endocarditis is shown by the appearance of a soft aortic diastolic murmur. In the second stage the degree of enlargement may be slightly greater, especially when there is mitral or aortic incompetence

Third stage. The physical signs are those of a chronic valvular lesion, the typical presystolic murmur of mitral stenosis develops, or the signs of aortic regurgitation, or both (see pp 216 and 218)

Diagnosis.—If accompanying acute rheumatism, the important thing is to recognise the existence of carditis by repeated examination of the heart and search for the foregoing signs Pallor in a patient with acute rheumatism is often an indication that carditis is present Failure of the temperature to respond to adequate dosage of salicylates means either that carditis is present or that the diagnosis is wrong Persistence of tachycardia after the temperature has become normal, particularly if the sleeping pulse rate is increased, usually means carditis, but a normal pulse rate does not exclude carditis

If occu[illegible]

[illegible] dist[illegible]

by th[illegible] [illegible], absence of splenic enlargement, negative blood culture, and tendency to heal instead of to progress

Signs similar to those met in the first stage of rheumatic *carditis may be met with in persons with chronic infections* (e.g. tuberculosis, chronic pyelitis), with other acute infections, with anaemia, or with thyrotoxicosis. In nervous patients with a rapid pulse and an innocent murmur the diagnosis may be difficult at first, but these patients have no fever, no cardiac enlargement, and the first sound is not soft. Some of them have circulatory symptoms ("effort syndrome", see p. 435) which are apt to cause suspicion to fall on the heart, *especially if the physician is unfamiliar with the syndrome*; but the concomitant signs and symptoms of the primary disease should prevent errors.

Course.—If the physical signs are those of the first stage only, complete resolution is not only possible but frequent, though some of these patients who have apparently made a complete recovery retain silent myocardial lesions which may cause auricular fibrillation or cardiac failure many years later. Once second stage signs are reached, recovery is usually not complete, but the patient is left with some degree of permanent cardiac damage. Recrudescences and relapses during con-*valescence are not infrequent, and are just as likely to be* accompanied by fresh cardiac damage as the initial attack. Second or third attacks after an interval of a few months or even a few years are also common.

In the majority of cases recovery from an acute attack results in a healed stationary lesion, if any lesion is left at all. In a few cases the endocarditis becomes chronic, and the lesion progresses very slowly over a period of years. In a small number of cases which start as a simple rheumatic endocarditis, fever persists, the process is progressive, the blood culture becomes positive, and signs of emboli appear, that is to say, *the condition starts as a simple endocarditis but a streptococcal* infection becomes superimposed at an early stage, and the condition develops imperceptibly into a subacute or an acute bacterial endocarditis *It is not uncommon for a subacute* endocarditis to become superimposed on a chronic endocarditis or on a healed stationary lesion after a lapse of years

Treatment.—The treatment of rheumatic endocarditis is described in detail on page 242. It may be summarised as follows: Complete rest in bed for as long as any evidence of activity remains; subsequently graduated and supervised convalescence.

Chronic Endocarditis and Healed Lesions.—The symptoms and physical signs are discussed later under the heading of Chronic Valvular Disease.

Subacute Bacterial Endocarditis

This is due to infection by *Streptococcus viridans*. The source of infection is probably the mouth or throat (dental sepsis or tonsillar sepsis). It is more common in males than females, and particularly in young adults (20 to 40). It tends specially to affect persons in whom one or more valves have previously been damaged by a simple rheumatic endocarditis; and it sometimes affects persons with congenital lesions; indeed, some physicians go so far as to say that it never develops in a person with an undamaged heart, and to diagnose a congenitally bicuspid aortic valve if subacute bacterial endocarditis appears in the aortic valve of an individual who has previously been examined and found to be healthy. Personally I doubt whether such a sweeping view is justified.

On the basis of histological studies, MacIlwaine has recently suggested that in subacute bacterial endocarditis *the valve lesions themselves tend to heal, but are prevented from doing so by repeated reinfection from a focus in relation to the teeth*. This view is the reverse of that hitherto accepted, namely, that the bacteriaemia in subacute bacterial endocarditis results from persistence and multiplication of the organisms in the vegetations, with constant reinfection of the blood stream. Nevertheless MacIlwaine's view is in better accord with recent therapeutic experience, both with sulphonamides and with penicillin, and it has important implications in connection with completion of treatment by discovery and eradication of the primary focus. One of my own cases speaks strongly in favour of MacIlwaine's view; this patient had her infection controlled by a course of penicillin extending over three weeks; a year later she developed an empyema of the left maxillary antrum, from the pus a strain of *S. viridans* was recovered which proved identical with that found in her blood a year previously; her heart became reinfected, and on this occasion penicillin proved unavailing.

Pathology.—Large cauliflower-like vegetations develop on the surface of the valve exposed to the blood stream. They

Signs similar to those met in the first stage of rheumatic carditis may be met with in persons with chronic infections (e.g. tuberculosis, chronic pyelitis), with other acute infections, with anaemia, or with thyrotoxicosis. In nervous patients with a rapid pulse and an innocent murmur the diagnosis may be difficult at first, but these patients have no fever, no cardiac enlargement, and the first sound is not soft. Some of them have circulatory symptoms ("effort syndrome", see p. 435) which are apt to cause suspicion to fall on the heart, especially if the physician is unfamiliar with the syndrome, but the concomitant signs and symptoms of the primary disease should prevent errors

Course.—If the physical signs are those of the first stage only, complete resolution is not only possible but frequent, though some of these patients who have apparently made a complete recovery retain silent myocardial lesions which may cause auricular fibrillation or cardiac failure many years later. Once second stage signs are reached, recovery is usually not complete, but the patient is left with some degree of permanent cardiac damage Recrudescences and relapses during convalescence are not infrequent, and are just as likely to be accompanied by fresh cardiac damage as the initial attack. Second or third attacks after an interval of a few months or even a few years are also common

In the majority of cases recovery from an acute attack results in a healed stationary lesion, if any lesion is left at all. In a few cases the endocarditis becomes chronic, and the lesion progresses very slowly over a period of years In a small number of cases which start as a simple rheumatic endocarditis, fever persists, the process is progressive, the blood culture becomes positive, and signs of emboli appear, that is to say, the condition starts as a simple endocarditis but a streptococcal infection becomes superimposed at an early stage, and the condition develops imperceptibly into a subacute or an acute bacterial endocarditis. It is not uncommon for a subacute endocarditis to become superimposed on a chronic endocarditis or on a healed stationary lesion after a lapse of years

Treatment.—The treatment of rheumatic endocarditis is described in detail on page 242. It may be summarised as follows: Complete rest in bed for as long as any evidence of activity remains; subsequently graduated and supervised convalescence.

or toes, on the thenar or hypothenar eminences, less often on the skin in other parts. A spot develops suddenly, and is spontaneously painful and acutely tender at first; it usually lasts only a short time (24-48 hours) and then disappears. Spots may appear at frequent intervals or only occasionally; often a patient will develop one or more spots daily for a few days, then none for a week or two. (2) Embolic nephritis is characterised by the appearance of blood and albumin in the urine. There is usually no rise of blood pressure, no oedema, and no rise of blood urea. Occasionally, however, a diffuse nephritis occurs instead of a focal nephritis, and there is raised blood pressure, oedema, and raised blood urea in addition to albuminuria and haematuria. (3) Small circular retinal haemorrhages. (4) Less constantly, gross infarcts in spleen, kidney, or brain, or in right-sided cases, in lungs.

The symptoms are usually slowly progressive, though periods of pyrexia may alternate with afebrile periods; the patient gradually goes downhill and dies in anything from three months up to two years, occasionally they live as long as five years, with periods during which the disease seems quiescent. Death usually results from the toxaemia, or from embolism. Many patients die without having developed congestive failure; but sometimes congestive failure appears a few days or a few weeks before death, especially when the subacute ulcerative endocarditis complicates old valvular disease.

Physical Signs.—(1) *There are signs of an endocarditis which is progressive*, the signs are the same as in rheumatic endocarditis, but the disease spreads from one valve to another, murmurs appear and disappear as vegetations develop or are broken off, and the heart tends to become progressively larger.

(2) *Enlarged spleen.*

(3) *Fever and anaemia.* The type of fever is very variable. Some cases show a continuous irregular fever; others have remittent or intermittent fever with profuse sweats; in a third group febrile and afebrile periods alternate. Occasionally patients are afebrile throughout the illness. The anaemia is of moderate severity, the haemoglobin level being frequently between 80 and 50 per cent, it is usually orthochromic and normocytic, and is very refractory to treatment. The leucocyte count is raised in some cases, normal in others; occasionally there is a mononucleosis. Many of the patients develop a

tend to spread over the valve cusp and to the wall of the auricle or ventricle. In the case of the mitral valve, infection is often conveyed to the septal wall of the left ventricle, and vegetations develop on the part touched by the septal cusp of the valve when it opens. The vegetations are easily broken off, and form emboli; when this occurs the surface of the cusp is left ulcerated; and occasionally perforation of a cusp occurs. Emboli are often very small and these minute emboli produce characteristic lesions in the skin ("Osler's spots") and in the kidneys ("embolic focal nephritis" or "flea-bitten kidneys"). Emboli also occur elsewhere, though less constantly, and infarcts may be found in the spleen, kidneys, liver, or brain; occasionally embolism of a limb occurs The endocarditis affects the valves of the left side much more often than those of the right side. When it affects the valves of the right side, or when it affects a congenital communication between right and left sides such as a patent septum or patent ductus arteriosus, the infarcts occur in the lungs

Hypertrophy and dilatation of the chambers are found corresponding to the valve or valves affected There is a localised septic myocarditis in the area immediately subjacent to mural vegetations, the myocardium there is infiltrated with leucocytes. The spleen is usually enlarged and firm, it may show infarcts as well

Symptoms.—There may be a history of former acute rheumatism from which the patient has made a good recovery, or which has left him with some degree of impairment of his cardiac reserve. Occasionally no history of previous rheumatism is obtained. In most cases the illness starts insidiously with symptoms pointing to a general infection; there is progressive weakness and pallor, malaise, loss of appetite, and irregular fever or an evening rise of temperature with a tendency to sweat at night. After a time the patient becomes breathless on exertion, or if he has previously suffered from breathlessness on exertion, it becomes worse and is brought on by a lesser amount of effort. The fingers become clubbed. Then symptoms of emboli appear. In a few cases emboli provide the first evidence of illness and the general febrile symptoms follow. The most frequent embolic manifestations are

(1) Osler's nodes—small tender red spots of about pin-head size, sometimes slightly elevated, which occur about the fingers

was easily sterilised, but bacteriaemia recurred on cessation of treatment

Immediately prior to the introduction of penicillin, my assistants, Drs. McLeod and Halcrow, were experimenting with continuous sulphonamide therapy. There were indications that this form of treatment was capable of arresting the disease in some cases, of 7 cases, 1 was apparently cured (observed for six months prior to his repatriation to Italy, 1 was temporarily controlled for four months, after which reinfection and death occurred, 1 was controlled for a few weeks, and 4 were complete failures. I believe that this treatment may yet prove of value in combination with penicillin in cases where the latter alone fails to arrest the infection. The method is to give the sulphonamide in full doses at first, if and when the temperature is controlled, dosage is reduced to ½ gm three or four times daily, and this is kept up for three months or more, a careful watch must be kept on the blood count as well as on the heart and temperature If the sulphonamide first tried fails to control the temperature, other members of the group should be tried.

The introduction of penicillin has completely altered the outlook in subacute bacterial endocarditis Though still a serious disease, the mortality has been reduced from 100 per cent to about 40 per cent. It has been clearly shown that the important point is to give large doses over a prolonged period. It seems to matter little whether the organism is penicillin-sensitive or penicillin-resistant *in vitro*, the earlier failures are now attributed to inadequate dosage The Penicillin Clinical Trials Committee recommend a dose of 500,000 units daily for a minimum of 28 days arrest of the infection (at least temporary) is claimed in 60 per cent of a series of 147 cases. The daily dose may be given in three equal parts at eight-hourly intervals, by intramuscular injection. Larger doses, up to 2,000,000 units daily, will sometimes arrest the infection when the standard dosage fails, aureomycin or chloromycetin may succeed

For bacterial [illegible]
arteriosus, surgical [illegible]
Chemotherapy or [illegible]
[illegible] as well, though arrest has been obtained in a number of cases from ligature alone Results from chemotherapy alone have been uniformly hopeless, penicillin alone has yet to receive adequate trial.

characteristic muddy brownish pallor described as a "café au lait" complexion.

(4) *Signs of emboli*. Osler's spots, embolic nephritis, retinal haemorrhages, infarcts of spleen, kidney, brain, limb, or (R-sided cases) lung.

(5) *Positive blood culture*, the organism being *S. viridans*. It should be noted that culture is not constantly positive throughout the disease; it may be positive early, then negative for a long time, then positive again; a single negative culture, or even several negative cultures in succession do not exclude a subacute bacterial endocarditis.

Course.—The disease is progressive, and fatal in from three months to five years. Until recently very few recoveries had been reported, and in most of them the diagnosis was not free from doubt: the mortality was generally regarded as being, to all intents and purposes, 100 per cent. Since the introduction of penicillin and of continuous sulphonamide therapy, an increasing number of arrested cases has been reported. Even in the most favourable circumstances, however, the disease still carries a mortality of something like 40 per cent.

Diagnosis.—The general symptoms and fever may suggest tuberculosis or other infection, or complaint of weakness might lead to a mistaken diagnosis of neurosis or anaemia. Careful examination of the heart will usually indicate an organic and progressive lesion, though in some cases the cardiac physical signs are delayed in developing. The presence of fever should prevent a mistaken diagnosis of splenic anaemia. The diagnosis from simple endocarditis depends on the progressive nature of the lesion, the splenic enlargement, and the positive blood culture. The same features plus the presence of fever distinguishes the condition from an anaemia simulating valvular disease.

Treatment.—The patient should be put to bed and given rest and general symptomatic treatment. Many methods of eradicating the infection have been tried in the past with little success: antiseptics, such as mercurochrome by injection; vaccines; sera; exposure to X-rays. The introduction of sulphonamides was hailed with hope but gave disappointing results; the combination of sulphonamides with heparin in the hope of preventing further deposition of fibrin on the valve cusps met with equally little success. In most cases the blood

malaria, etc. The leucocytosis and the positive blood culture with discovery of a cardiac lesion clinch the diagnosis.

Embolic type. There are symptoms of embolism in some situation—brain, spleen, kidney, lung, or limb—followed by continued fever, appearance of a cardiac lesion, and the other symptoms enumerated above. The *diagnosis* depends on recognising the original lesion as embolic, and then seeking the source of embolism. Fibrillation, coronary thrombosis, and aortic aneurysm are easily excluded; the distinction between embolism and thrombosis may be more difficult.

Cardiac type. There are symptoms of progressive cardiac failure—first dyspnoea on exertion becoming more severe, then oedema becoming more extensive—associated with fever, enlarged spleen, and positive blood culture; emboli may occur during the course of the illness. This type is especially likely when malignant endocarditis develops as a complication of some chronic cardiac lesion. *Diagnosis*: When a patient with a cardiac lesion develops progressive cardiac failure with a normal rhythm, but with fever, the possibility of ulcerative endocarditis should be investigated.

Insidious type. The symptoms are the same as in subacute bacterial endocarditis, but the progress is more rapid, and death occurs in from three to four weeks up to about six months. For *diagnosis* see Subacute Bacterial Endocarditis.

Prognosis.—The disease is invariably fatal if untreated, the duration varying from a few weeks (even one week) up to three or four or occasionally six months. Recently recoveries have been recorded following sulphonamide or penicillin.

Treatment.—See Subacute Bacterial Endocarditis, the remarks regarding which apply with equal force. The prospects of success are about equal in the two conditions.

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Once subacute bacterial endocarditis has been arrested by one or other of the foregoing methods, it would appear to be important to seek the primary focus of infection and to eradicate this. A wise precaution is to give penicillin for 48 hours before and after any surgical procedure, especially dental extraction. A similar precaution is amply justified in any patient with a rheumatic cardiac lesion.

Acute Ulcerative or Malignant Endocarditis

Malignant endocarditis differs from subacute bacterial endocarditis mainly in degree. It is secondary to infection elsewhere, and various organisms are concerned, including haemolytic and other streptococci, staphylococci, pneumococci, meningococci, typhoid bacilli, *H. influenzae*, etc.

Pathology.—The appearances vary somewhat according to organism concerned. Staphylococcal vegetations resemble those of *Streptococcus viridans*, but the kidney shows pyaemic abscesses instead of embolic focal nephritis, and these are also found in prostate and bones; the spleen is usually soft and diffluent. Other streptococci tend to produce mainly ulceration of valve cusps with small vegetations, and sometimes perforation of cusps. Pneumococci may produce tough fibrinous vegetations, sometimes long shreds of fibrin hanging into ventricle or auricle; the spleen is small and firm. The remainder resemble staphylococcal endocarditis.

Symptoms.—These again differ from subacute bacterial endocarditis mainly in degree, being more acute, more sudden in onset, and more rapidly progressive. Various clinical types are described:

Typhoid type The symptoms are prolonged fever, continuous or intermittent, with progressive weakness, malaise, anorexia; later restlessness or delirium, then the patient sinks into a semi-comatose muttering delirium ("typhoid state"), and finally dies in coma. A cardiac lesion develops during the course of the illness, but may not be obvious for some time after the onset. There is a high leucocytosis, and the spleen is enlarged in most cases. Death occurs in from one to five weeks. The *differential diagnosis* is from other illness causing continued fever—typhoid, paratyphoid, miliary tuberculosis, septicaemia, pyaemia, abortus fever, malta fever,

CHAPTER 9

CHRONIC ENDOCARDITIS AND HEALED VALVULAR LESIONS

General Considerations.—The most common cause of organic damage to valve cusps is *acute endocarditis*; if the patient survives, damage to the cusps often persists in the shape of scarring and deformity (chronic valvular disease). In a small percentage of cases a low-grade, smouldering inflammatory process persists (true *chronic endocarditis*), and the valve lesion becomes progressively more severe, an acute exacerbation may supervene at any time Note that it is strictly incorrect to refer to chronic healed lesions as "chronic endocarditis" this term should be reserved for the slowly progressive, active cases

The aortic valve may also be damaged by pathological processes spreading from the aorta, especially *syphilis* *Atheromatous deposits* are occasionally found on the mitral valve; it is doubtful whether these interfere with the function of the

Congenital lesions of valves are considerably less frequent. The most common is stenosis of the pulmonary valve, a developmental error, aortic stenosis also occurs as a developmental error, but stenosis of the aorta at the level of the ductus arteriosus (coarctation of the aorta) is more frequent Foetal endocarditis is another possible cause of congenital valve lesions, but is probably rare

Trauma is a very occasional cause of valve lesions. The aortic valve cusps may be ruptured from a blow on the chest wall, or from sudden strain raising the blood pressure, especially in persons with arteriosclerosis Penetrating wounds have been known to damage valve cusps.

Apart from the above conditions, in which there is organic damage to the ...

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heard at the aortic area, conducted to the vessels of the neck, and sometimes to the descending thoracic aorta or even the abdominal aorta. The pulse is of small volume and anacrotic.

Diagnosis.—Aortic systolic murmurs are very common while aortic stenosis is comparatively rare; aortic stenosis should not be diagnosed from a systolic murmur alone, but only if the murmur is accompanied by a thrill and by signs of left ventricular hypertrophy.

Aortic Regurgitation

Causes.—(1) Acute endocarditis and (2) damage from former endocarditis. (3) Syphilitic aortitis. Some twenty years ago syphilis was the most common cause, but it was becoming steadily less common, and by 1939 was much less frequent than rheumatism. During the war there was a very considerable increase in the incidence of syphilis while movements of a population tended to interfere with adequate treatment; an increase in the frequency of syphilitic lesions of the aortic valve may be expected within the next few years. (4) Exceptionally aortic regurgitation is secondary to a dilated aorta in hypertension, and in this case it may be transient.

Effects.—Blood regurgitates from the aorta into the left ventricle during diastole, so that (a) the diastolic blood pressure falls, and (b) the left ventricle is dilated since it receives its normal quota of blood from the auricle plus that which regurgitates from the aorta. The left ventricle then hypertrophies, and compensation is effected by hypertrophy and dilatation of the left ventricle. The output of the left ventricle is now greater than normal, as it includes the blood which will regurgitate during the next diastole plus the normal output which will flow onwards. The increased output is associated with a greater rise in pressure during systole; thus while the diastolic pressure falls, the systolic usually rises slightly and the pulse pressure is increased—a BP of 120/80 is converted to one of 130/50, or 140/60.

Symptoms.—If compensation is adequate and the lesion is stationary, there are no symptoms, and the patient can lead a normal life. There may be any grade of impaired cardiac reserve, from breathlessness on severe effort only, through breathlessness on moderate effort, to established fail

"valvular disease" or as "incompetence of such-and-such a valve"; but the diagnosis should be that of the primary condition which has led to the dilatation. The *incompetence* is temporary, disappears when its cause is removed, and often it makes no difference to the patient's circulation, any inefficiency of which is due to the primary disease; it makes no difference to the prognosis or treatment. The causes of functional valvular lesions have been fully discussed in the section on Irrelevant Cardiac Murmurs (see pp. 56 and 57).

Damage to valve cusps may lead to regurgitation through the valve; or the cusps may become thickened and adherent, or even calcified, so as to obstruct the blood flow (stenosis). At the mitral valve, stenosis is more common than simple incompetence—mitral incompetence is much more often functional than organic; at the remaining valves, incompetence is more common than stenosis.

Aortic Stenosis

Aortic stenosis of mild to moderate grade is not infrequent in cases of aortic regurgitation (q.v.); but in these cases the features of the incompetence usually overshadow those of the stenosis. More severe grades of aortic stenosis with little or no incompetence are relatively uncommon. Endocarditis (rheumatic or arrested subacute bacterial) is the usual cause, but some cases in older patients result from calcification of the aortic valve secondary to arteriosclerosis or atheromatosis. When aortic stenosis results from endocarditis, mitral stenosis often co-exists.

Effects.—Compensation is effected by concentric hypertrophy of the left ventricle.

Symptoms.—There may be none if compensation is adequate, or any grade of impairment of cardiac reserve up to established cardiac failure. The latter is usually left ventricular in type; faintness, dizziness, breathlessness, palpitation, or syncopal attacks are frequent in the early stages. Rarely aortic stenosis produces the Bernheim syndrome (see p. 290).

Physical Signs.—There is a forcible heaving apex impulse, displaced slightly to the left, a systolic thrill is palpable at the aortic area (2nd right interspace), the cardiac dullness is slightly enlarged *to the left*; *a coarse systolic murmur* is

while the stream from the auricle impinges on the other side (Austin Flint murmur). The pulse is of large volume but ill sustained, the pulse waves rising quickly and falling abruptly; it is described as a "water-hammer pulse" from the resemblance to the sensation felt on inverting the children's toy of that name (The "water-hammer" is a closed glass cylinder containing a small quantity of water and evacuated of air.) The water-hammer pulse is best appreciated by laying the flat of the fingers across the patient's wrist while elevating his arm; it is also strikingly demonstrated by laying the hand across the dorsum of the foot, when it can be felt in the dorsalis pedis artery. The blood pressure is characteristic, the systolic being normal or raised, the diastolic lowered, and the pulse pressure high, sometimes, on attempting to take the blood pressure, the diastolic cannot be estimated at all, a sharp sound continuing right down to 0 mm. This sound ("pistol-shot sound") can often be heard on listening over the femoral arteries.

X-ray in aortic regurgitation shows marked prominence of the left ventricular contour with enlargement to the left and downwards (Fig 11, p 86) A cardiogram usually shows left axial deviation unless mitral stenosis coexists, in advanced cases T1 becomes inverted and is regarded as a sign of left ventricular hypertrophy

Auricular fibrillation is relatively uncommon as a complication of aortic regurgitation; when it does occur it is due to associated mitral stenosis, to associated myocarditis, or to myocardial fibrosis

Mitral Stenosis

Mitral stenosis is the result of simple (rheumatic) endocarditis It rarely results from a single attack, except where this has given rise to a true chronic endocarditis, but more often follows repeated attacks It may develop in cases of subacute rheumatism, or in patients who have had one or more attacks of chorea, even though there has been no sign of acute endocarditis during the actual attack of chorea. Occasionally mitral stenosis is found in persons who deny having had acute rheumatism, tonsillitis, subacute rheumatism or ...

symptoms even at rest. The earlier symptoms are often referable *to deficient output into the aorta or to the high pulse pressure*. throbbing in the neck or head, tinnitus (patients can occasionally hear their own murmurs), dizziness or actual fainting, specks before the eyes, breathlessness on exertion, and palpitation; there may be angina of effort, since the lowered diastolic pressure may prevent adequate filling of the coronary vessels. Patients often have a pale complexion, even though blood examination shows no anaemia. Later, with more advanced grades of failure, a functional regurgitation at the mitral valve may occur, and symptoms of pulmonary congestion develop—cough and spit, perhaps cyanosis, occasionally pulmonary oedema. Still later, symptoms of congestive failure may appear—distended veins, enlarged liver, and oedema; but many patients with aortic regurgitation die before this stage of failure is reached. With advanced failure in aortic valvular disease, patients sometimes complain of a sensation of levitation, i e that they are floating off the bed —*presumably a symptom of cerebral anaemia. Cheyne-Stokes* respiration may also occur

Physical Signs.—The earliest physical sign is an aortic diastolic murmur, and this may be *the only sign* for a considerable time In advanced cases there is usually pallor. Arterial pulsation is visible in the neck (as opposed to the visible venous pulsation of congestive failure). The apex impulse is forcible and heaving; often the whole chest shows heaving pulsation. The apex is displaced downwards and outwards, often to the 6th or 7th space beyond the mid-clavicular line The cardiac dullness is correspondingly enlarged A diastolic murmur is audible in the 2nd right space, conducted down the left border of the sternum and also towards the apex: the murmur is sometimes best heard, or heard only at lower end of sternum; if difficult to hear, it can be intensified by examining the patient sitting up and leaning forwards If the diastolic murmur is conducted to the apex, as it approaches that point it may be intensified during the period of auricular systole, producing a presystolic murmur; in many cases this is due to mitral stenosis occurring along with the aortic regurgitation; but in some there is no mitral stenosis, and the presystolic accentuation is thought to be due to the regurgitant stream of blood impinging on one side of the septal cusp of the mitral valve

terminates in an abrupt slapping first sound. Another frequent murmur, which tends to occur earlier in the development of the condition, is a mid diastolic murmur at the apex. Occasionally these two murmurs are continuous. The third murmur sometimes heard in mitral stenosis is a pulmonary diastolic murmur due to dilatation of the pulmonary artery (Graham Steell's murmur). In addition to the murmurs there is a characteristic slapping first sound, and there is marked accentuation of the second sound at the pulmonic area.

These physical signs are altered if there is mitral regurgitation as well as stenosis; the heart is then enlarged to the left, and a systolic murmur is heard at the apex as well as the presystolic

The presystolic murmur of mitral stenosis is due to auricular systole forcing blood through the narrowed mitral valve, and it disappears under certain conditions (1) If the auricles cease to contract as co-ordinate chambers, viz. in auricular fibrillation and auricular flutter. (2) If the auricles and ventricles contract simultaneously, as in nodal rhythm, nodal extrasystoles, and with some auricular extrasystoles. (3) If auricular contraction is separated by an interval from ventricular contraction (latent block) the murmur ceases to be presystolic and becomes mid diastolic. (4) If the auricular contractions are weak and ineffective, even though the rhythm is normal this occurs with gross dilatation and failure of the left auricle, and with marked tachycardia. Thus, immediately after an attack of acute pulmonary oedema, the presystolic murmur is often inaudible, and it may remain inaudible for a day or two

The typical presystolic murmur is characteristic, but mistakes are made frequently a common error is to regard either a coarse systolic murmur, or a split first sound, as a presystolic murmur The presystolic murmur may be overlooked, as it is strictly localised to a point just internal to the apex If in doubt the murmur can often be brought out by making the patient lie on his left side after taking mild exercise and ...

... one of the most frequent causes of a breakdown in compensation Its onset is sometimes preceded by auricular extrasystoles for some time

the orifice, and in such circumstances the signs of obstruction may disappear if the vegetation is broken off as an embolus. Except for the rare cases of "relative" and "functional" mitral stenosis there are no other causes; syphilis is never a cause of mitral stenosis.

Effects.—The left auricle dilates and hypertrophies. This alone may compensate cases in which the degree of obstruction is slight, but it is rarely sufficient with any considerable degree of obstruction. In such circumstances the pulmonary circulation is congested, the pressure in the pulmonary artery is raised, and the right ventricle hypertrophies. Compensation is thus effected by the left auricle and the right ventricle, and it involves permanent congestion of the pulmonary circulation. *Note that the left ventricle plays no part in compensating a pure mitral stenosis*, only when the valve is incompetent as well as stenosed does the left ventricle hypertrophy.

Symptoms.—With adequate compensation there are none. There may be any grade of impaired reserve from breathlessness on severe effort only, up to established cardiac failure. The earlier symptoms usually have reference to pulmonary congestion—palpitation with breathlessness, cough and spit, cyanosis. Occasionally attacks of acute pulmonary oedema occur. In the later stages or in more advanced cases the symptoms are those of congestive failure with oedema, cyanosis, distended veins, enlarged liver. Auricular fibrillation commonly develops in the later stages, and infarcts may complicate the symptomatology.

Physical Signs.—In early cases a presystolic murmur at the mitral area may be the only physical sign. In more advanced cases there is frequently a characteristic appearance or "mitral facies", i e a malar flush which may be slightly cyanotic, set on a faintly yellowish background (due to bilirubinaemia); the lips are highly coloured and often cyanosed.

The apex impulse is normal in position; it is often "slapping" in character, and preceded by a *presystolic thrill*. The cardiac dullness is not enlarged, but in the late stages when the right auricle is dilated and failing there may be enlargement to the right. Epigastric pulsation from the hypertrophied right ventricle is frequently visible.

The characteristic murmur is the presystolic murmur, which starts faintly in the later half of diastole, is crescendo, and

early cases both X-ray and cardiograms may be negative, the presystolic murmur being the only sign of the condition.

Mitral Regurgitation

This was formerly a very common diagnosis as cases of "functional" mitral regurgitation were included; but the vast majority of cases of organic damage to the mitral cusps involve either stenosis alone, or regurgitation with a greater or less degree of stenosis, and they are nowadays labelled "mitral stenosis". "Pure" mitral regurgitation without any stenosis is relatively uncommon as an organic lesion. It is the result of past or present endocarditis affecting the mitral valve.

Effects.—When the left ventricle contracts, some blood passes to the aorta and some regurgitates to the left auricle. The left auricle meantime receives the usual quota from the pulmonary circulation, and therefore becomes dilated; it later hypertrophies. When now the left auricle contracts, it delivers this additional quantity of blood to the left ventricle, which in turn dilates, then hypertrophies. Compensation is thus brought about by hypertrophy of the left auricle and left ventricle. It differs from mitral stenosis in that the left ventricle and not the right is involved, and in that full compensation can be achieved without congestion of the pulmonary circulation.

Symptoms.—There may be none, or any grade of impairment of reserve up to established cardiac failure. The earlier symptoms are palpitation and breathlessness; there may be symptoms of deficient output into the aorta, or of pulmonary congestion.

Physical Signs.—The appearance is not characteristic, patients may be cyanosed in later stages. The apex impulse is forcible and the apex displaced to the left, there may be a palpable systolic thrill. The cardiac dulness is correspondingly enlarged. A systolic murmur is audible at the apex, and usually conducted to the axilla, sometimes to the angle of the left scapula. The murmur is usually high-pitched and blowing, it tends to replace rather than accompany *the first* sound.

Diagnosis.—Systolic murmurs at the mitral area are very common, mitral regurgitation is not diagnosed from finding a systolic murmur alone—there must be signs of hypertrophy

X-ray in mitral stenosis shows enlargement of the pulmonary conus and left auricular appendix; this produces a filling-up of the left middle arc which becomes convex instead of concave in the A–P view; the enlarged conus is well seen in the right oblique view, which also shows backward displacement of the barium—filled oesophagus by the enlarged left auricle (Figs. 6 to 9, pp. 80 to 84). Exceptionally in mitral

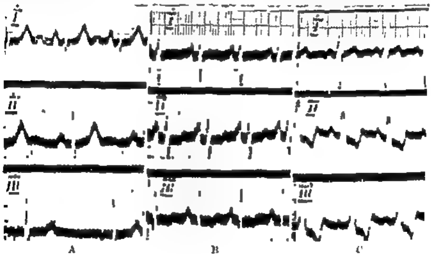

FIG 57.—Mitral stenosis

A. *Mitral stenosis* P large and pointed in lead 1, slightly notched in lead 2, and diphasic in lead 3 Right axial deviation RT iso-electric and T upright in all leads

B. P large, pointed, and upright in all leads Right axial deviation RT slightly depressed *and T diphasic in leads 2 and 3, indicating commencing right ventricular strain*

C

stenosis the left auricle remains of normal size while the pulmonary artery and right ventricle are enlarged, one such case, verified at autopsy, led me to alter a clinical diagnosis of mitral stenosis to one of congenital heart disease, the pathologist was unable to determine the reason for the failure of the left auricle to enlarge in this case of extreme mitral stenosis. Cardiograms in mild degrees of stenosis are normal or may show slight enlargement and notching of the P waves In advanced cases P is large, notched, and broad *in leads 1 and 2, often diphasic* in lead 3; the ventricular complexes show well-marked right axial deviation; T3 may be inverted and Q3 prominent. In

occurs as a sequel to mitral stenosis (Graham Steell murmur). Endocarditis of the pulmonary valve is a rare cause.

Effects.—The right ventricle becomes dilated and hypertrophied, the process corresponding exactly to what happens with the left ventricle in aortic regurgitation.

Physical Signs.—There is epigastric pulsation from right ventricular hypertrophy and perhaps enlargement of cardiac dulness to the right (if the right auricle is dilated). A diastolic murmur is audible to the left of the sternum and is conducted downwards.

Diagnosis.—There may be difficulty in distinguishing a pulmonary diastolic from an aortic diastolic murmur; patients are more likely to be cyanosed than pale, there is no capillary pulsation and no water-hammer pulse; the pulse pressure and blood pressure do not show the characteristic features of aortic regurgitation X-ray shows right ventricular enlargement, and the cardiogram usually shows right axial deviation.

Tricuspid Stenosis

Tricuspid stenosis, though infrequent, is not so rare as it is sometimes thought to be In the majority of cases there is mitral stenosis and sometimes aortic stenosis as well. The lesion is the result of endocarditis.

Effects.—There is dilatation and hypertrophy of the right auricle, which is the only chamber available to compensate the lesion

Symptoms.—Although *adequate* compensation is less likely than with other valvular lesions, it is surprising how long the patients can carry on before they develop failure; in fact the presence of tricuspid stenosis in addition to mitral stenosis seems to delay the onset of pulmonary congestion The earl

symptoms and even in the advanced stages of failure there is *often a striking absence of orth*opnoea, the patient being able to lie quite flat despite gross cyanosis, oedema, and hepatic enlargement

Physical Signs.—The veins of the neck are distended and often show pulsation The liver is usually greatly enlarged.

of the left ventricle as well; and even then, a relative incompetence secondary to hypertension, aortic VDH, or myocardial disease must be excluded before one assumes an organic mitral valve lesion as the explanation. Note that mitral regurgitation may occur (as shown by post-mortem findings) when no systolic murmur has been audible during life; in fact it is stated that in half the patients with a mitral systolic murmur there is no regurgitation, and in half those with regurgitation there is no systolic murmur. Radiologically, there is enlargement of the left ventricle and left auricle. Cardiograms may show P waves similar to those of mitral stenosis, but left axial deviation instead of right; the findings are not constant.

Pulmonary Stenosis

Most cases are congenital. Infrequently it arises as an acquired lesion from the results of endocarditis. In congenital cases there is usually a patent intraventricular septum and often patent ductus arteriosus; without these the condition is liable to be incompatible with life.

Effects.—The lesion causes hypertrophy of the right ventricle.

Symptoms are usually well marked, consisting of breathlessness and cyanosis, progressing to congestive failure.

Signs.—Marked cyanosis and clubbing of fingers are present. Epigastric pulsation (right ventricular) is visible. There is a systolic thrill at the pulmonary area. The cardiac dullness is enlarged to the right. A coarse systolic murmur is audible at the pulmonic area, conducted upwards and outwards.

Diagnosis.—Pulmonary systolic murmurs are very common, while pulmonary stenosis is relatively uncommon; it should be diagnosed only if the murmur is accompanied by a thrill and signs of right-sided hypertrophy, cyanosis, and clubbing. Radiologically there is enlargement of the right ventricle and pulmonary conus. Cardiograms usually show gross right axial deviation.

Pulmonary Regurgitation

Pulmonary regurgitation is not common. Occasionally a functional regurgitation secondary to pulmonary hypertension

Course of Chronic Valvular Disease

As already indicated, two types of case can be differentiated by their history and their course. In a small group, assumed to have a genuine chronic endocarditis, the lesion is slowly but steadily progressive, terminating in failure within two or three years. There is slow but steady deterioration in the cardiac reserve with gradually increasing enlargement of the appropriate cardiac chambers The accepted criteria of activity for acute cases, viz. temperature, sleeping pulse rate, and blood sedimentation rate are usually negative, despite which the downhill trend persists

In the second and much larger group, the course is considerably longer, often amounting to 20 years and sometimes to 40 years or more. It is characterised by prolonged periods during which the lesion is apparently absolutely stationary. During the earlier years the most frequent cause of a breakdown is a fresh attack of acute rheumatism or tonsillitis, intercurrent infections such as coryza, influenza, bronchitis, or pneumonia also appear to be capable of "re-activating" the quiescent lesion, yet many patients escape fresh rheumatic infection, and many others pass apparently unscathed through the intercurrent infections During the later years, and especially in mitral stenosis, auricular fibrillation is apt to develop; it is the most frequent cause of a breakdown in long-standing cases of mitral stenosis. In addition to the likelihood of its causing congestive failure, it involves the risk of embolism. A complication which may arise at any stage and in either type of case is subacute bacterial endocarditis; this danger appears to be greatest in those with oral sepsis, especially just after dental extractions

The Management and Treatment of Chronic Valvular Disease

The first step in dealing with a case is to make sure that the lesion is quiescent and stationary. This is usually made evident by a careful history, but in some cases a period of observation is required during which temperature, sleeping pulse rate, physical signs, and the blood sedimentation rate are watched. Secondly, the patient's cardiac reserve must be

A presystolic thrill may be felt at the lower edge of the sternum; the cardiac dullness is enlarged to the right; a presystolic murmur similar to that of mitral stenosis is sometimes heard just to the left of the lower end of the sternum. In a number of proved cases, however, the murmur has been systolic only. The presystolic murmur may disappear under the same conditions as that of mitral stenosis. The signs of mitral stenosis are usually present as well.

Diagnosis.—Disproportionate enlargement of the liver with striking absence of orthopnoea in a case of cardiac failure suggests either tricuspid stenosis or chronic pericarditis (*pericarditic pseudo-cirrhosis of liver*). *The characteristic tricuspid* murmur should be sought. X-ray shows right auricular enlargement. Cardiograms are not characteristic, but may show large P waves in leads 2 and 3, with a low or flat PI, resembling the P waves of emphysema.

Tricuspid Regurgitation

Tricuspid regurgitation is rare as an organic lesion due to endocarditis; but "functional" regurgitation occurs in conditions analogous to those under which functional mitral regurgitation is found.

Effects.—*The right auricle and right ventricle become hypertrophied*, the process being analogous to that which occurs with left auricle and left ventricle in cases of mitral regurgitation.

Symptoms.—There may be none, or any grade of impaired reserve.

Signs.—There is cyanosis, venous pulsation in the distended neck veins, and epigastric pulsation from an engorged liver. The apex impulse is normal. The cardiac dulness is enlarged to the right. A systolic murmur can be heard at the base of the sternum.

Diagnosis.—The same remarks apply as in the case of mitral regurgitation. Organic tricuspid regurgitation is diagnosed only after "innocent" and "irrelevant" murmurs have been excluded.

Congenital Lesions

The murmurs of congenital lesions are sometimes mistaken for valvular disease; the symptoms and signs are described in Chapter 19.

If extractions are required, it is inadvisable to remove more than one or two teeth at a sitting; sulphonamide or penicillin (preferably the latter) should be given for 48 hours before and after the operation.

When there is established cardiac failure, the treatment consists of rest and digitalis; note that digitalis is contra-indicated in the absence of cardiac failure or auricular fibrillation. The treatment of cardiac failure is described in Chapter 22, and the treatment of auricular fibrillation has already been discussed on pp 138 to 140

In the case of aortic valve lesions due to syphilis, the question of antisyphilitic treatment arises. If the syphilis is recent or active, it should be treated with iodides followed by mercury and bismuth Neosalvarsan and its allies are dangerous in these cases and should never be given to a patient with active syphilitic aortitis If the syphilis is of old standing and stationary, antisyphilitic treatment will make no difference; the damage is already done It should be remembered that it is the patient who is to be treated, not the Wassermann reaction.

In all cases the patient's general health should be carefully reviewed, and any concomitant condition such as anaemia, etc, should be treated His habits with regard to alcohol and tobacco should also be considered, while these need not be forbidden, moderation should be enjoined.

The surgical treatment of mitral stenosis (mitral valvulotomy). Between 1920 and 1930 a number of attempts were made to dilate stenosed mitral valves surgically, but success was exceptional and the attempts [illegible]

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[illegible] this country, in America and in France Some surgeons have made a venous shunt by anastomosing the azygos vein to a branch of the inferior pulmonary vein, some have created a septal defect with the object of reducing the high pressure in the pulmonary circulation More spectacular, however is the operation of mitral valvulotomy as practised by Brock of London, and Bailey, Glover and O'Neill of Philadelphia, the stenosed valve is incised or split by passing a finger into the left auricle Brock reports nine cases with seven successes, the American team report twenty-two cases with eleven survivals The limited experience of the operation at present available suggests that

assessed; this also is best achieved by a careful history, assuming the patient is honest in his replies.

If the lesion is stationary and compensation is adequate, no treatment is required. The patient may lead a normal life, though it is not advisable that he should engage in occupations involving sustained physical stress such as coal-carrying, etc.; less severe forms of manual labour are not contra-indicated with adequate compensation. Competitive athletics are inadvisable though games need not be forbidden; I have seen a number of young men with valvular lesions who have assured me that they played Association or Rugby football regularly and who have shown no sign of being any the worse of this activity.

When there is impairment of the cardiac reserve, treatment is a matter of adjusting the patient's activities and habits to his cardiac capacity. Each case must be judged in relation to his individual capacity, activities which cause undue fatigue or undue breathlessness in that particular patient should be forbidden, all other activities may be allowed. It is a great mistake to permit a patient to be more restricted by his doctor than by his disease. I have no hesitation in stating that the patients are much better to be employed than to be on the sick list; if a patient's normal occupation is unsuitable, the sooner he is found suitable employment the better. The patients should be advised to have eight to nine hours in bed each night, they should take time over their meals and should avoid rushing immediately afterwards. It is important to prevent the development of an anxiety neurosis by giving the patient the best prognosis that is justifiable rather than the worst, and by carefully avoiding any suggestion of permanent invalidism as long as the condition is not totally incapacitating. It is also important to avoid sore throats and acute respiratory tract infections as far as possible, patients should be discouraged from frequenting overcrowded and ill-ventilated places, and they should not knowingly expose themselves to infection. Should an acute throat or respiratory infection develop, rest in bed must be enforced, and a careful watch kept for signs of activity of the lesion during the subsequent two weeks; in the case of a tonsillitis, salicylates should be given to cases of rheumatic origin. Oral hygiene is of considerable importance in the prevention of subacute bacterial endocarditis, and regular dental attention should be advised.

SECTION 3. THE AETIOLOGICAL VARIETIES OF HEART DISEASE

THE main diseases which affect the heart and may cause heart lesions are as follows:

INFECTIONS.—Acute rheumatism. Syphilis. Acute and sub-acute bacterial endocarditis.

Diphtheria is in a different category, as the myocarditis caused resolves if the patient recovers, and diphtheria does not leave permanent damage.

HYPERTENSION.—Hypertensive heart disease.

ARTERIOSCLEROSIS.—Coronary disease: coronary sclerosis, coronary thrombosis.

CHRONIC LUNG DISEASE.—Pulmonary heart disease or "cor pulmonale".

ENDOCRINE DISORDERS.—Thyrotoxicosis—"thyrotoxic heart disease". Myxoedema—"myxoedema heart".

DEFICIENCY DISEASES.—Vitamin B_1 deficiency—"beri-beri heart".

CONGENITAL LESIONS.

TRAUMA.

NEUROSIS.—Neurocirculatory asthenia. Cardiac neurosis. Effort syndrome.

Many physicians classify heart disease in this way—e.g. rheumatic or syphilitic, etc.; it has advantages both from the point of view of prognosis and of treatment, and in this section heart disease is described from this angle.

CHAPTER 10

ACUTE RHEUMATISM AND ALLIED DISEASES

Rheumatic fever is a disease of extreme importance because of its maiming effect on the heart. Though it does not often kill during the acute stages, it is responsible for much invalidism and for many deaths later in life, and it is extremely common in this country.

Note that acute rheumatism is a specific disease, quite

the ideal case is one with a tight mitral stenosis, little or no mitral regurgitation, little or no enlargement of the left ventricle, and symptoms referable mainly to pulmonary hypertension; this is a well defined group, though it forms only a small proportion of the total cases of mitral stenosis. Contra-indications include active rheumatism, serious involvement of other valves, left ventricular enlargement, extensive calcification of the mitral valve, secondary sclerotic changes in the pulmonary arteries, long-standing auricular fibrillation, and long-standing right-sided heart failure.

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effusions or from the heart valves, though recently claims to have grown it from heart valves have been made.

Owing to the failure to isolate the specific haemolytic streptococcus from the most frequent and most serious lesions (heart valves), alternative theories have been advanced. Attempts have been made to blame a filter-passing virus for the disease, regarding the streptococcus as a purely accidental finding, but the evidence so far is less convincing than that in favour of a streptococcus.

Many attacks of acute rheumatism occur at a short interval after an acute sore throat, viz. from twelve to twenty-five days later. In a ward with convalescents, an epidemic of sore throats is often followed by an epidemic of relapses. This fact has led many to believe that the attacks of polyarthritis and the endocarditis are allergic reactions to a streptococcal throat infection, the streptococcus involved being the haemolytic streptococcus. This would explain the ease with which the organism can be cultivated from the throat, the difficulty of cultivating it from the heart valves and joint effusions, and the occurrence of the attack after a sore throat. This is the most widely held view of acute rheumatism at present, but it should be remembered that it is merely a tentative view, not proven as yet, and indeed there is some evidence against it though there is much in favour.

Three factors, then, seem to be involved in acute rheumatism—a constitutional susceptibility, a haemolytic streptococcal infection (possibly a virus infection), and an allergic reaction to the infection.

Pathology

There is nothing specific about the *tonsils*—many show evidence of chronic infection, but many are normal in the intervals between attacks. The *joint lesions* consist of a synovitis with effusion into the cavity of a slightly turbid fluid containing shreds of fibrin and leucocytes. The most important changes are in the heart—a *myocarditis* characterised by the presence of Aschoff's nodes (collections of endothelial cells some of which are multinucleate, with polymorphs, lymphocytes, and often eosinophils; they are found chiefly in the sub-endocardial and perivascular zones); when they heal they leave minute fibrous scars, often demonstrable only with diffi-

distinct from the motley group of conditions described as chronic rheumatism by lay persons and by some doctors.

AETIOLOGY

The disease affects both sexes in almost equal proportions. It is especially common in children, and becomes less frequent as age advances; it is most common between 5 and 15, few cases occur below 5; after 15 it becomes progressively less frequent, until after 40 it is again rare. Nevertheless, I have seen a case at 63. In children, the heart rarely escapes, in adults it escapes much more often

The disease sometimes occurs in apparently healthy children, but more often affects debilitated, delicate, or weakly children. It is more common in the children of the working classes, the children of the well-to-do are less apt to develop typical rheumatic fever, but frequently have rheumatic carditis without joint manifestations. Diet has been thought responsible for this—lack of calcium and lack of vitamin A. Damp climate and damp houses predispose. Several members of one family are often affected. The disease has a very striking geographical distribution, it is endemic and very common in this country; even here, different towns are affected in a different degree; and even in the one town, the incidence may vary considerably in different districts

The actual cause of acute rheumatism is by no means settled as yet. Formerly it was thought to be a constitutional disease. When the infective factor was recognised, the constitutional factor became neglected but the tendency recently has been to lay more stress on the constitutional factor again, and many of the points noted above regarding the incidence of the disease emphasise the importance of constitution or "susceptibility." The very striking tendency for attacks to recur at intervals in a given patient further illustrates the importance of susceptibility.

Early this century Poynton and Payne claimed to have isolated a specific diplococcus, now thought to be a streptococcus. A haemolytic streptococcus can be cultivated from the throat in the acute stage of most cases: it has occasionally been cultivated from subcutaneous nodules and from the blood in fatal cases; but it can rarely be obtained from the joint

effusions or from the heart valves, though recently claims to have grown it from heart valves have been made.

Owing to the failure to isolate the specific haemolytic streptococcus from the most frequent and most serious lesions (heart valves), alternative theories have been advanced. Attempts have been made to blame a filter-passing virus for the disease, regarding the streptococcus as a purely accidental finding; but the evidence so far is less convincing than that in favour of a streptococcus.

Many attacks of acute rheumatism occur at a short interval after an acute sore throat, viz. from twelve to twenty-five days later In a ward with convalescents, an epidemic of sore throats is often followed by an epidemic of relapses. This fact has led many to believe that the attacks of polyarthritis and the endocarditis are allergic reactions to a streptococcal throat infection, the streptococcus involved being the haemolytic streptococcus. This would explain the ease with which the organism can be cultivated from the throat, the difficulty of cultivating it from the heart valves and joint effusions, and the occurrence of the attack after a sore throat This is the most widely held view of acute rheumatism at present, but it should be remembered that it is merely a tentative view, not proven as yet, and indeed there is some evidence against it though there is much in favour

Three factors, then, seem to be involved in acute rheumatism—a constitutional susceptibility, a haemolytic streptococcal infection (possibly a virus infection), and an allergic reaction to the infection

Pathology

There is nothing specific about the *tonsils*—many show evidence of chronic infection, but many are normal in the intervals between attacks *The joint lesions* consist of a synovitis with effusion into the cavity of a slightly turbid fluid containing shreds of fibrin and leucocytes. The most important changes are in the heart—a *myocarditis* characterised by the presence of Aschoff's nodes (collections of endothelial cells some of which are multinucleate, with polymorphs, lymphocytes, and often eosinophils, they are found chiefly in the subendocardial and perivascular zones), when they heal they leave minute fibrous scars, often demonstrable only with diffi-

culty, but occasionally sufficient to produce an interstitial fibrosis. Occasionally some twigs of the coronary vessels show an acute arteritis with similar nodes in their walls. The *endocarditis* has been described—warty vegetations, later scarring and deformity of the cusps, perhaps mitral stenosis, aortic regurgitation or both. The *pericarditis* has also been described—either a fibrinous pericarditis or a serous effusion. The *myocardium is involved in nearly every case in children*, the endocardium in many, the pericardium in a smaller number; in a good many cases the disease is a pan-carditis. *Rheumatic nodules* are subcutaneous nodules with a structure similar to the Aschoff nodes of the myocardium, they resemble a con*glomeration of such nodes.*

Less frequent lesions in rheumatism are found in the *brain* (cases of chorea along with the somewhat rare rheumatic encephalitis); nodes occur similar to Aschoff nodes along with perivascular infiltration. *Pleurisy* is not very uncommon, *pneumonia* much less frequent; it is an interstitial pneumonia in which Aschoff nodes have been demonstrated. *Nephritis* is very rare, but even in the kidneys Aschoff nodes have been reported.

Clinical Features

Acute rheumatism is met with in a variety of clinical forms. It may be described under the following headings:—

(1) The acute attack of rheumatic fever.
  (a) Without carditis.
  (b) With carditis
  (c) With carditis and other complications.
(2) Acute carditis without joint manifestations.
(3) Subacute rheumatism
(4) Subacute carditis
(5) Chorea.

Acute Rheumatic Fever.—The attack may occur in a previously healthy individual, or in one who has been "delicate" and suffered from some of the symptoms to be described under the heading of Subacute Rheumatism. In many cases the attack develops from twelve to twenty-four days after a sore throat; the sore throat is often quite mild, and the patient may not mention it unless asked specifically, a few patients deny

having had a sore throat, and in a few the sore throat and the joint pains are simultaneous.

At the onset of the attack, the patient complains of pain, swelling, and stiffness in one or more joints; he has a rise of temperature and pulse rate, loss of appetite, and general malaise. The affected joints are swollen from effusion, hot and tender; they are usually normal in colour, but occasionally show a faint pink or red blush. In any one joint, pain and swelling last a day or two if untreated, but as one joint is improving others are becoming affected, so that the pains "flit about from joint to joint." The larger joints are particularly affected—especially knees, elbows, wrists, and ankles, less often hips or shoulders, occasionally the small joints of the hands or feet.

If untreated, the pain and swelling continue to come and go for ten days or more, then gradually subside and the temperature returns to normal. If treated with adequate doses of salicylates, the fever and joint pains are abolished in 24-48 hours, but they return if the salicylates are stopped in less than ten days. In the vast majority of cases the joint symptoms clear up entirely and leave no after-effects; in occasional cases a large joint such as the shoulder remains painful and stiff for some weeks afterwards, resembling an acute or subacute infective arthritis, in occasional cases an illness starts like acute rheumatism, but the small joints of the hand become involved, the swelling becomes periarticular and persistent, and the case progresses as an acute rheumatoid arthritis. Many physicians try to draw a hard-and-fast line between acute rheumatism and acute rheumatoid arthritis, insisting that they are entirely separate entities, and that these "intermediate cases" are cases in which diagnosis has been mistaken; but I have seen a typical acute rheumatism with carditis progress to the rheumatoid type, and I am convinced not only that this intermediate type does occur but that it is by no means infrequent. This is in accord with the fact that typical rheumatic cardiac lesions are sometimes found in patients with chronic rheumatoid arthritis.

Acute Rheumatic Fever with Carditis.—In many cases of carditis there are no [illegible]

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carditis. In some cases there is pericardial pain; or there may be acute dyspnoea from myocarditis. Symptoms of congestive failure are very rare in the acute stage.

The physical signs of carditis have already been described in detail (Chapters 7 and 8)

Features which suggest that carditis is present, and which should lead to a particularly close watch on the heart are:

Pallor —Patients without carditis are often flushed; patients with carditis more often pale. *Temperature*:—Failure of the temperature to fall to normal in 24-48 hours when adequate doses of salicylates are given usually means either that carditis is present, or that the diagnosis is wrong (e.g. acute rheumatoid arthritis, osteomyelitis, etc.) *Tachycardia*:—Persistent elevation of pulse rate after the temperature has reached normal is often due to carditis, especially if the sleeping pulse is elevated (some nervous children show an increase in pulse rate as soon as it is counted). Note, however, that a normal pulse rate does not exclude carditis, especially if salicylates are being administered. *Abnormalities of rhythm*:—In many cases there is a latent block demonstrable only by electrocardiograms; but in some this may give rise to occasional dropped beats. Occasional cases show transient auricular fibrillation. Even the sudden appearance of numerous extrasystoles should be regarded with suspicion. *Nodules*:—Subcutaneous nodules are rare in the absence of carditis—and usually imply a serious carditis. They are not present in all cases—indeed there are years in which nodules are seen in almost every case, and years in which they are scarcely seen at all. They are small, shotty nodules, palpable underneath the skin and freely movable, slightly tender or not at all so. They are found especially on the extensor surfaces of the elbows and forearms, knees and legs, over the extensor aspects of the knuckles, over the occiput, and sometimes over the spine. *Sedimentation rate* —The blood sedimentation rate is raised in acute rheumatism, but usually returns to normal when the acute manifestations pass off, failure to return to normal when other acute manifestations have disappeared suggests a possible carditis. *General condition* —Anorexia or a fickle appetite persisting after the acute symptoms have subsided is suspicious of active carditis. Anaemia which fails to improve with iron likewise points to active infection.

Acute Rheumatic Fever with Other Complications.—*Skin eruptions*, though infrequent, are occasionally met with. *Sudamina* and *miliaria* sometimes result from profuse sweating in the acute stage. *Urticarial* or *erythematous* rashes may appear, possibly from drugs used in treatment. Eruptions which have led to considerable controversy are *erythema nodosum* and *purpura*. Formerly regarded as rheumatic, *erythema nodosum* is now known to be a manifestation of tuberculosis in the vast majority of cases, in a few instances it is thought to be rheumatic, while in exceptional cases some other infection such as chronic meningococcal septicaemia is regarded as responsible. The eruption is due to sensitivity to a specific bacterial toxin, most often tuberculin, occasionally haemolytic streptococcal toxin, rarely some other toxin. In rheumatic cases it is not often seen during the acute attack; it may precede this or occur in an interval between attacks. The eruption consists of bright pink, raised, tender swellings, especially in the legs over the tibiae. *Purpura* is sometimes accompanied by a flitting synovitis (the group of cases formerly termed "peliosis rheumatica"). Some physicians maintain that this condition is quite distinct from rheumatic fever, others allege that carditis may be present and that the disease is in fact, acute rheumatism complicated by purpura. One of my cases had carditis, polysynovitis, a purpuric eruption, and large haemorrhages into the periarticular tissues of the elbows and knees: the nature of the carditis was proved at autopsy. Such an occurrence might be explained either as an allergic reaction to drugs of the salicylate group, or as a manifestation of sensitivity to a streptococcal throat infection. Indeed, when current views regarding the aetiology of acute rheumatism and of toxic purpura are compared it is surprising that a combination of the two is so infrequent.

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tinged pink; and signs of consolidation in one or both lungs. Some deny the existence of rheumatic pneumonia, maintaining that these symptoms are due (a) to atelectasis from pressure of a pericardial effusion, or (b) to infarction. Nevertheless the clinical features differ from both these conditions; they appear to correspond to what Hadfield has described and I myself have seen post mortem. The mortality is high in cases with rheumatic pneumonia; some say that it is invariably fatal, but this is not true; I have seen some seven cases with two deaths and five recoveries.

Peritonitis and other abdominal complications. Abdominal pain is not uncommon at the onset of rheumatic carditis. Cases sometimes find their way into a surgical ward labelled "appendicitis"; an innocent appendix may be removed, or the label may be altered to "ileo-caecal" or "mesenteric lymphadenitis", in either case, after a few days during which pyrexia has persisted, the true diagnosis becomes apparent with the appearance of painful swollen joints or of unequivocal signs of carditis. In some of these cases the abdominal pain is no doubt a referred pain due to involvement of the diaphragmatic pericardium; in others there may perhaps be a genuine abdominal lymphadenitis during the stage of onset. A third possibility is a true *rheumatic peritonitis*, as in the following example:

A boy of 12 had joint pains with fever and an endocardial murmur for a few days before he developed severe abdominal pain. A surgeon of considerable experience, who was fully alive to the presence of active carditis, diagnosed peritonitis and performed a laparotomy with much misgiving. The appendix and remaining abdominal organs were healthy, but the retro-peritoneal tissue, especially in the perinephric regions, was the seat of an intense inflammatory reaction with blood-stained serous exudate. The abdomen was closed and the subsequent course of the illness was that of a rheumatic pan-carditis with pericardial effusion; after a few months the boy recovered, being left with an apparently stationary aortic regurgitation. A year later he again became ill with symptoms of intestinal obstruction; at operation, three loops of bowel were strangulated by a fibrous band and were excised. On the following day he had fever and pericarditis, while polysynovitis developed a few days later; again the subsequent course was that of an acute rheumatic carditis. It is noteworthy that the fever and joint pains responded to salicylates but not to sulphonamides on each occasion.

Nephritis is a rare complication. I have seen a haemorrhagic nephritis without oedema; also diffuse nephritis with oedema and hypertension.

Chorea is very rare during an acute attack, though attacks of chorea and of acute rheumatism frequently occur at different times in the same patient; but if a child suffering from chorea develops acute rheumatism or acute endocarditis, the chorea usually disappears Occasionally mild twitching movements persist

Encephalitis This complication is not described in the standard text-books, but I have seen two cases—one characterised by drowsiness and confusion with squint and amaurosis, the other by violent convulsive movements and delirium. The former recovered, the latter was proved at post mortem.

Hyperpyrexia was frequent in pre-salicylate days, but has been rare since the introduction of salicylates. I have only seen one case, a young man of 20, with a typical rheumatic fever; despite salicylates, on the eighth day of illness he suddenly became thirsty, then delirious, and within six hours his temperature reached 108° F, he died half an hour later.

Acute Carditis without Joint Manifestations.—Rheumatic carditis may follow an attack of tonsillitis in the absence of joint pains, sometimes it occurs without any definite history even of tonsillitis These cases are by no means infrequent, and they are important as the diagnosis is apt to be more difficult The children of the well-to-do seem to be more liable to carditis without joint pains, whereas the poor more often have typical joint pains and swelling In some cases there are *no joint or muscle pains whatever*, in others there are vague fleeting pains in the muscles or joints, with perhaps a little stiffness but no effusion, and they may receive scant attention from parents, being regarded as "merely growing pains" All intermediate grades between classical rheumatic polysynovitis and complete absence of arthritic manifestations are met with in rheumatic carditis. In the absence of joint pains the symptoms are fever, pallor, anorexia, malaise, and rapid pulse, with signs of pericarditis, of endocarditis, or of both, these have been described in detail in Chapters 7 and 8. Abdominal pain is not infrequent in this group of cases

Subacute Rheumatism.—Many children, on recovering from an attack of acute rheumatism, do not regain perfect health,

but suffer for a time (it may be for months) from vague ill health. Other children may suffer from identical symptoms without ever having an acute attack, and they may, in the course of time, develop a cardiac lesion indistinguishable from one which has resulted from acute rheumatism.

The symptoms of this state of ill health, known as subacute rheumatism, are as follows. The children are pale and weakly; they tend to be languid, preferring to sit over the fire than to go out and play; appetite is poor or fickle—they pick at their meals instead of enjoying them. They complain from time to time of vague fleeting pains in the muscles or joints. They are liable to recurring sore throats. The pulse is usually elevated (90-100) and from time to time there may be a slight rise of temperature in the evenings (99°-100°); with this there may be night sweats. If the child also has enlarged tonsils and adenoids causing a cough, the resemblance to early tuberculosis may be very close. The other condition which may give identical symptoms is chronic pyelitis, which should always be excluded by microscopic examination of the urine. Children with subacute rheumatism are liable to develop an acute attack at any time, and they may, in the course of time, develop mitral stenosis or other cardiac lesion.

Subacute Carditis.—In addition to the symptoms of subacute rheumatism there may be complaint of breathlessness. Often the apex impulse is diffuse, extending beyond the nipple line, a soft systolic murmur is heard at the apex, and the second pulmonic sound is accentuated These physical signs may disappear completely after 24 hours' rest in bed, but reappear when the child is allowed up—even after long periods of rest in bed. They probably indicate a myocarditis Finally there are cases in which carditis is chronic from the start and completely symptomless The individuals concerned are regarded as healthy; they have a good attendance record at school, taking part in games and other activities; the cardiac lesion develops insidiously and is found accidentally at a much later date, often on examination for entry to the Forces, or for Life Insurance; by this time there is established mitral stenosis or aortic regurgitation. Sometimes the lesion is first discovered when failure develops in middle age in a patient who gives no history of illness or of symptoms which could be recognised as an acute or subacute stage of the disease.

Chorea (Sydenham's chorea—not to be confused with Huntington's chorea).—This usually affects children, aged 5 to 20, more often girls than boys. Rheumatic infection is a *sine qua non*; but the chorea either precedes or follows the rheumatism, they are rarely present together. Children with a family history of nervous disease or who are themselves emotionally unstable are predisposed to chorea rather than polyarthritis. Signs of endocarditis are uncommon in first attacks, less uncommon in subsequent attacks, but many cases who have shown no signs of endocarditis during any of their attacks are later found to have developed mitral stenosis. It is curious that aortic regurgitation is uncommon as a sequel to chorea.

The onset is usually insidious, the child is fretful, easily tired, sleeps badly, and is irritable and nervous. Then she becomes fidgety, clumsy, and may drop things or fall. The typical features develop in a week or two, namely *choreiform movements*. These are irregular, jerky, purposeless, and inco-ordinate movements affecting face, limbs, and shoulders; the same movement is not often repeated twice in succession. Associated there are weakness, sometimes mental changes, and sometimes inco-ordination of voluntary movement. The movements are more marked when the patient is watched, and they cease during sleep. They may be so violent as to prevent swallowing or to cause the patient to damage herself.

The physical signs, in addition to the typical movements, are inco-ordination in voluntary movements; ill-sustained irregular grasp, the tongue is protruded and withdrawn jerkily with a flop. There is diminished muscle tone, the outstretched hands droop at the wrist while the fingers are hyperextended. The reflexes are unaltered, which allows distinction from Friedreich's ataxia in which similar movements may occur. Delirium is rare. There is no fever.

The condition lasts from six weeks up to several months. Second attacks after an interval of a few months are common. Death is rare, it may occur from exhaustion, occasionally from a cardiac complication.

The diagnosis is from tics in which the same movement is repeated time and again, from Friedreich's ataxia in which the knee-jerks are lost and plantar responses are extensor; and from encephalitis in which there are usually some eye changes, the onset is sudden and somnolence is common.

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stage, these lesions usually disappear without leaving permanent after effects.

DIAGNOSIS

Polyarthritis must be distinguished from osteomyelitis and pyogenic arthritis in which a single joint is affected, the skin is more red and glazed, and there is deep oedema; the disease does not flit from joint to joint, and the patient is more acutely ill, with high leucocytosis, and possibly rigors. Acute rheumatoid arthritis affects multiple joints, the small joints of the hands and feet are commonly involved, the swelling is much more persistent in any one joint, and the response to salicylates is less. Gonorrhoeal arthritis resembles rheumatoid arthritis, but tends to affect larger joints, the swelling is more peri-articular and more persistent than in rheumatic fever; it tends to leave stiff joints. Polyarthritis sometimes occurs in typhoid, dysentery, influenza, and pneumonia during convalescence, but the history gives the diagnosis. Scurvy produces tender swellings which are subperiosteal. Purpuras and haemophilia may be associated with joint swelling or with haemorrhage into joints. Tuberculous joints are much more chronic and usually single. Clutton's joints occur in congenital syphilitics.

Endocarditis and myocarditis have to be recognised when present, and to be distinguished from (a) functional conditions (innocent murmurs, neurocirculatory asthenia, cardiac neurosis), (b) old stationary valve lesions, (c) syphilis in the case of the aortic valve, (d) acute and subacute bacterial endocarditis

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Subacute rheumatism must be differentiated from early tuberculosis, and from focal sepsis, especially chronic pyelitis or bacilluria. "Growing pains" should not be accepted as a diagnosis but a thorough local and general examination should be carried out to determine their cause.

The diagnosis of *chorea* from tics, Friedreich's ataxia, and encephalitis has been discussed.

TREATMENT OF ACUTE RHEUMATISM

Acute Attack.—Rest in bed is essential. In a child with a first attack, as a rule a minimum of six weeks *should* be insisted

A single acute attack with little or no cardiac damage may end in complete recovery and never return. More often, attacks recur after an interval of months or years, or rheumatic fever and chorea may alternate with one another, there may or may not be symptoms of subacute rheumatism in the interval. Cardiac damage is equally likely in first or subsequent attacks.

Rheumatic heart disease can be considered in three stages, viz. an acute stage of *active carditis* (Chapters 7 and 8), a *healed stationary stage* (Chapter 9), and a *stage of cardiac failure* (Chapters 1 and 2). Many cases pass through these three distinct stages which may cover a span of 20, 30, or even 40 years. It is important to remember that the *acute stage* is not necessarily accompanied by pain and swelling in the joints. In most cases it heals with the production of a stationary lesion, but in a few it results in a genuine chronic endocarditis with a slowly worsening valvular lesion, while in some others it fails to heal and passes directly into the stage of failure within a few months. The symptoms in the active stage are sometimes subacute rather than acute. Finally there are cases in which no acute stage can be recognised; a patient who is unaware of having had any illness and who has a record of perfect school attendance is found to have an established rheumatic valvular lesion. The *stationary stage* may remain so for many years, or may be punctuated by periods during which there is temporary recrudescence of activity. In the more fortunate patients compensation is adequate, and this stage is attended by little or no disability; in their less fortunate brethren the cardiac reserve is impaired with a corresponding degree of disability. The *stage of failure*, again, is sometimes interrupted by periods of improvement or temporary recovery, maintained for a few months or a few years, but ultimately the failure recurs and the disease claims its victim.

The joint manifestations usually leave no permanent effects; occasionally a joint remains stiff (acute infective arthritis), and occasionally rheumatic fever is followed by an arthritis of rheumatoid type. In most cases the nervous symptoms of chorea clear up entirely, but children may remain nervous and jumpy for some time. Pulmonary and nephritic lesions are generally found only in the more serious cases of carditis, a number of whom die; but if the patient survives the acute

It is generally accepted that salicylates have no effect in arresting carditis: nevertheless they abolish the joint pains, thereby adding to the patient's comfort; and in some cases they lower the temperature and pulse rate, lessening the work of the heart and assisting the provision of rest. There is no good evidence that salicylates are ever harmful in carditis, and no valid reason for withholding them. Digitalis is contra-indicated, it may convert a latent into a high-grade block, and it may produce alarming symptoms; it is never of benefit in the absence of cardiac failure, and, in the rare cases in which cardiac failure complicates the active stage of carditis, digitalis is usually ineffective For the pain of pericarditis, counter-irritation should be tried, if this fails to give relief, morphine may be given to an adult or nepenthe to a child. The indications for paracentesis of a pericardial effusion have been discussed on p 104

When all evidence of activity has disappeared, gentle exercise should be started while the patient is still in bed. After another few days the patient is allowed up, on the first day for fifteen minutes then for lengthening periods each day After a day or two he is allowed to walk a few steps, and from this point he is put on graduated exercise

Chorea is treated by rest, which must be both physical and mental All excitement must be barred and the patient's room must be kept quiet, if treated in a general ward she should be screened off during the acute stage In violent cases a cot or cot-bed may be necessary to prevent her falling out, and the sides should be padded Artificial feeding is occasionally needed in severe cases, this can be carried out through a catheter passed through the nose into the oesophagus. Salicylates or aspirin in dosage similar to that employed in acute rheumatic fever usually produce marked benefit, personally I consider aspirin the more efficacious of the two in chorea, 5 grains may be given thrice daily to children aged 5 to 10, and 10 grains thrice daily to older children Some physicians prefer sedatives and order luminal, prominal, or chloral hydrate · $\frac{1}{2}$ to $1\frac{1}{2}$ grains of luminal can be given thrice daily according to age. In some cases both aspirin and a sedative are required The minimum period of rest for a first attack should be six weeks, or longer if the choreiform movements have not entirely ceased at the end of that time

on. In adults, if there is no sign of active carditis and if there is a normal sedimentation and pulse rate, three weeks may suffice. Movement and vibration can be extremely painful—even a heavy step on the floor—and these should be diminished. In view of sweating, the nightgown should be of flannel, and patients are best nursed between blankets. It was formerly taught that they should be kept flat on their back, but this is probably unsound; they may be allowed any position which they find most comfortable. They must be kept quiet and resting—nursed, not allowed to make efforts themselves. During the febrile stage, diet should be light and nourishing; fluids may be allowed *ad lib.* Once fever has subsided full diet may be allowed.

Sodium salicylate is specific for the pains and temperature; it should be given in full doses with at least an equal quantity of sodium bicarbonate (many physicians give double the quantity of sodium bicarbonate). An adequate dose for most adults is 20 grains four-hourly, but if this is insufficient, the dosage may be 20 grains two-hourly, the patient being watched for deafness or tinnitus These doses should be kept up till the patient has been free from pain and fever for a day or two; then the dose may be reduced to 15 grains thrice daily and this should be continued for four to six weeks. Some patients react better to aspirin. Methyl salicylate is sometimes applied to the joints, but it is not necessary when full doses of the sodium salt are given by mouth.

Carditis necessitates continued rest as long as any evidence of activity persists. The period of rest varies from six or eight weeks up to several months. Criteria of activity include fever, changes in cardiac physical signs or size, presence of nodules, sleeping pulse rate, and blood sedimentation rate The PR interval of the cardiogram may also be of help Poor appetite, pallor in the absence of anaemia, and failure of anaemia to improve with iron are other features which suggest that the disease is still active It is fashionable at present to lay chief stress on the sleeping pulse rate and the blood sedimentation rate; it is necessary to remember that a normal sleeping pulse rate does not exclude activity in patients who are receiving large doses of salicylates, that cardiac failure lowers the blood sedimentation rate and may therefore mask activity, while anaemia raises the blood sedimentation rate and may thus simulate activity.

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Convalescence from acute rheumatism should be slow and prolonged, especially if there has been carditis. Children should be kept under careful supervision for a year or two. Most patients require iron for anaemia; cod liver oil and malt may perhaps diminish the liability to further attacks. Good diet, fresh air, sunlight, and outdoor exercise when fit for it are essential—in other words, a sanatorium regime. Meantime education should not be neglected, and a specified number of hours should be set apart for it each day. If a sore throat develops, the child should be kept in bed and watched for fourteen days, and salicylates should be given for three to four weeks after an apparently simple sore throat. Some authorities recommend the continuous use of sulphonamides in small doses ($\frac{1}{2}$ to 1 gram daily) as a prophylactic, personally I prefer penicillin applied as a paint to the tonsils. Convalescence from acute rheumatism is often associated with the development of flat-foot, unless this fact is borne in mind, the resulting complaint of pain may erroneously be attributed to a recurrence of the rheumatism. An attempt should be made to prevent the occurrence of flat-foot by appropriate exercises for the muscles of the feet and legs during the final stages of the period of rest in bed and the earlier stages of convalescence.

Subacute rheumatism is treated as described for convalescent cases, the children are kept under observation, confined to bed during periods of mild activity, and put on a "sanatorium regime" during periods of quiescence.

Tonsillectomy.—Some physicians advise tonsillectomy indiscriminately in rheumatic children This is quite wrong. Tonsillectomy will not prevent subsequent attacks; indeed it is sometimes followed immediately by an attack or by an exacerbation. *In no circumstances should tonsillectomy be permitted while there is any evidence of active carditis*; septic tonsils at such times may be painted with a glycerine paint containing 10,000 to 100,000 units of penicillin per c.c During quiescent periods tonsils may be removed, *but only if they are obviously septic and accompanied by persistent enlargement of the tonsillar glands.* Doubtful tonsils are better left *in situ*, and healthy tonsils are more likely to protect against the disease than to cause it. I am certain that the majority of tonsillectomies in rheumatic children are ill advised if not actually harmful In a very few cases tonsillectomy is really necessary and beneficial.

affects a particular segment it is called a "fusiform aneurysm". Alternatively, a localised portion of the wall is apt to become stretched and to form a localised bulge, or a pouch projecting from the main vessel, this is called a "saccular aneurysm". Thrombosis usually occurs in the sac of the aneurysm, which becomes filled with laminated clot; and to this extent strengthens the wall once more However, portions of the clot may become dislodged to form emboli, and infarcts result in distant organs Alternatively the aneurysmal sac may rupture despite the thrombus, leading to fatal haemorrhage, this is one of the common causes of death in aortic aneurysm. The rupture is sometimes external, after the aneurysm has eroded through ribs and/or sternum; more often the rupture is into a bronchus, into the pleura, into the oesophagus, or into the pericardium.

In many cases of syphilitic aortitis the heart becomes involved, and this often occurs long before the stage of aneurysm is reached In its spread from the ascending aorta towards the heart the syphilitic process encounters (1) the orifices of the coronary vessels, (2) the site of attachment of the aortic valve cusps, and (3) on further extension it reaches the interventricular septum where it produces a true syphilitic myocarditis Much depends on the chance distribution of the plaques, in one case coronary orifices are affected while valve cusps escape, in the next, valve cusps are involved and coronary orifices escape, in a third, both are affected.

Coronary Orifice Involvement.—A single plaque abutting on a coronary orifice produces a degree of narrowing If there are plaques on each side, the orifice may be narrowed to a mere slit between them All degrees of coronary orifice stenosis may be met with. The condition usually develops slowly, and having progressed up to a point, it may disappear with cicatrisation of the plaque, only to reappear as a fresh plaque develops Occasionally it develops rapidly; this is especially likely to occur in response to injection of organic arsenical preparations in an active case (particularly if a small dose is used, and especially in patients who have not had preliminary potassium iodide) Severe grades of coronary orifice stenosis cause *sudden death* if they develop rapidly; *or progressive cardiac failure followed by death* if they develop gradually. Lesser grades of stenosis give rise to *angina of effort* clinically, and pathologically

CHAPTER 11

CARDIO-AORTIC SYPHILIS

SYPHILIS affects the cardiovascular system in the late secondary and early tertiary stages. The symptoms appear from two to five years or more after the primary infection. Cardio-aortic syphilis is therefore rare below the age of 20; it becomes increasingly frequent up to the age of 25-30, and has its maximum incidence about 30-35; thereafter it becomes less frequent. Both sexes are affected.

PATHOLOGY

The process starts first in the ascending aorta and it extends proximally towards the heart, and distally as far as the level of the diaphragm (junction of thoracic and abdominal aorta); the abdominal aorta is not affected in most cases, but occasionally syphilis is responsible for aneurysms forming here also. The initial lesion is a *mesaortitis*, syphilitic granulation tissue (lymphocytes and plasma cells) invades the media along the course of the vasa vasorum. Gradually the granulation tissue is replaced by fibrous tissue. The net result, as far as the media is concerned, *is a gradual replacement of muscular and elastic* tissue by fibrous tissue, with consequent loss of elasticity and weakening of the wall. Secondary changes occur also in the intima; where an area of infiltration comes close to the intima, the latter *undergoes reactive hyperplasia and becomes raised* to form a so-called "syphilitic plaque". Recent plaques are circular or oval, slightly raised though they may have a small depression at their centre, and are pearly white in colour, they are quite unlike the irregular yellow lesions of atheroma. As cicatrisation occurs in the media, it affects the intimal plaques also, and they become shrunken and scar-like. The process is a slow one, and various stages are often seen side by side—fresh plaques appearing while earlier ones are in various stages of cicatrisation.

In consequence of the mesaortitis described above, the aortic wall becomes weakened and less elastic. There is usually some generalised dilatation. When this is more marked and

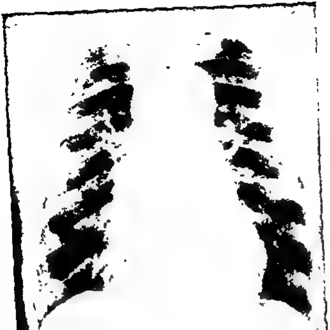
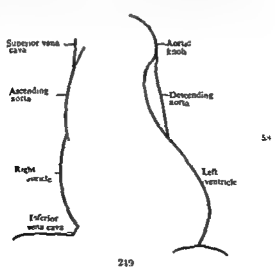


FIG 54 —Cardio-aortic syphilis. Note the wide aorta and enlarged left ventricle Male aged 60, complaining of pains from left axilla to sternum with tightness in chest, chiefly at night, of 1 month's duration Aortic diastolic murmur present BP 164/74 Electrocardiogram showed slight left axial deviation, otherwise normal Wassermann reaction + +.



they lead to para-arterial fibrosis identical with that which results from coronary atheroma (i.e. to a non-specific degenerative fibrosis of the myocardium). The angina may be followed *later by progressive cardiac failure. If stenosis of slight to* medium grade develops rapidly, the clinical picture may be that of *coronary occlusion*; and in fact thrombosis is apt to develop as a secondary effect in a vessel whose orifice is thus stenosed; the result will be a *myocardial infarct.*

Aortic Valve Involvement.—Syphilitic infiltration at the site of attachment of the aortic valve cusps is followed by distortion and thickening of the cusps themselves, resulting in *aortic regurgitation* In consequence of aortic valve involvement, the left ventricle undergoes hypertrophy. The clinical symptoms and signs do not differ from those of rheumatic aortic valvular disease, but anginal pain is present in a larger *proportion of cases and the electrocardiogram is more often* abnormal owing to the frequency with which coronary orifice stenosis coexists.

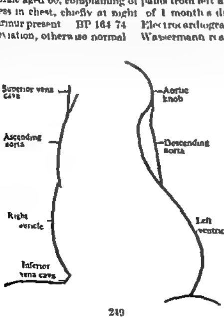
Further extension leads the syphilitic process into the interventricular septum, where a *genuine syphilitic myocarditis* may be found. *Very rarely*, cardio-aortic syphilis begins in the myocardium as a myocarditis, and affects the aorta only later if at all. In this region, the auriculo-ventricular bundle is particularly likely to be involved, leading to *heart block*. The myocarditis may also cause progressive deterioration of cardiac reserve leading to cardiac failure.

Note that syphilis does not affect the mitral valve. There may be dilatation of the mitral ring with relative incompetence and a mitral systolic murmur, secondary to aortic valve involvement, to coronary artery stenosis, or to genuine syphilitic myocarditis, but syphilis does not cause mitral *stenosis* Mitral stenosis in a syphilitic patient is due to independent rheumatic carditis, and is unconnected with the syphilis.

Symptoms and Signs

(1) **Early Stage of Syphilitic Aortitis.**—In this stage there are usually no subjective symptoms (these do not appear until later), but the condition may be recognised by the appearance of a loud ringing second sound at the aortic area, the result of dilatation of the aorta. There are only *two causes of a loud*

FIG 58.—Cardio-aortic syphilis. Note the wide aorta and enlarged left ventricle. Male aged 60, complaining of pains from left axilla to sternum with tightness in chest, chiefly at night, of 1 month's duration. Aortic diastolic murmur present. BP 164/74. Electrocardiogram showed slight left axial deviation, otherwise normal. Wassermann reaction + +



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8

ringing second aortic sound (*bruit de tambour*); they are hypertension (which is easily excluded by taking the blood pressure) and syphilitic aortitis or aneurysm. It is important to be on the look-out for this physical sign in any patient who is under treatment for syphilis, or who has had syphilis within the previous five years even though he is not under treatment

FIG. 59A—Cardio-aortic syphilis. Fusiform aneurysm of ascending aorta [illegible] descending aorta. Antero-posterior view. In [illegible], but [illegible]

at the time. It may be possible in such a case to demonstrate widening of the aortic shadow radiologically (Fig. 58). It is only rarely possible to demonstrate it clinically in the shape of increased dullness behind the manubrium sterni.

(2) **Coronary Stenosis.**—There may be angina of effort, or an attack of coronary occlusion. The symptoms and signs do not differ materially from those produced by coronary disease (Chapter 15). The patient is often younger and the ringing second sound may provide a clue.

(3) **Aortic Regurgitation.**—The symptoms and signs are identical with those of rheumatic aortic regurgitation, but the age at which they develop is usually later (rheumatic carditis has its maximum incidence between the ages of 5 and 15);

There may simply be breathlessness with a soft ... sound

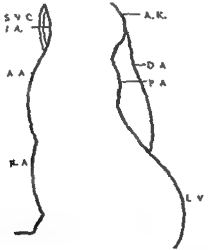

FIG 59A (key)

S V C = Superior vena cava	A K = Aortic knob
I A = Innominate artery	D A = Descending aorta
A A = Ascending aorta	P A = Pulmonary artery
R A = Right auricle	L V = Left ventricle

and an abnormal cardiogram, or there may be heart block. The age of onset is the most suggestive diagnostic feature.

(5) **Aneurysm.**—(a) *Ascending aorta* ("the aneurysm of physical signs"). There is often angina of effort from simultaneous coronary orifice stenosis, or there may be aortic regurgitation. Some patients complain of a more or less continuous pain in the upper sternal region from erosion of ribs or sternum; in others the pain is throbbing. Palpitation is a symptom in some cases. There is a systolic thrill over the second (or first and second) right interspace, pulsation and dullness on percussion in this area, with a harsh systolic murmur

conducted to the vessels of the neck. The differential diagnosis is from aortic stenosis (presence of pulsation and retrosternal dullness, while the heart is not enlarged unless there is aortic valve involvement). The aneurysm may be confined to the ascending aorta, or may involve part of the arch as well, in

FIG. 59B—Left oblique view The fusiform aneurysm is well seen in the ascending aorta lying above the heart shadow The remainder of the aorta is widened and tortuous

which case the signs and symptoms of aortic arch aneurysm are present *also. In advanced cases the aneurysm sometimes* erodes through the chest wall (ribs or sternum), appearing as a *pulsating swelling beneath the skin* (Fig. 60)

(b) *Arch of aorta* ("the aneurysm of symptoms") There is usually a harsh brassy cough from pressure on the left bronchus, and this becomes still more characteristic if there is left abductor laryngeal paralysis from pressure on the left recurrent laryngeal nerve; the cough then has a peculiar, hollow quality, and

there is hoarseness in addition to the cough. Many patients have difficulty in swallowing from pressure on the oesophagus. There is pulsation in the suprasternal notch, with tracheal tugging; if the fingers are placed below the larynx and the latter is pressed gently upwards, it is tugged downwards with each aortic pulsation. The radial pulses are apt to be unequal, and the blood pressure differs in the two arms. The

pupils are often unequal from pressure on the sympathetic. Left-sided abductor paralysis is demonstrable on laryngoscopic examination. The differential diagnosis is from bronchial carcinoma and other intrathoracic neoplasms, from enlarged tracheo-bronchial glands, and from a retrosternal goitre. X-ray screening is especially valuable for diagnosis, since it demonstrates the expansile pulsation of the aneur[illegible]

[illegible] in the aneurysmal sac lessens the pulsation and sometimes abolishes it. A tortuous aorta can be mistaken for an aneurysm on an X-ray film if only one view is taken and the patient is not screened.

(c) *Descending thoracic aorta.* This is the most difficult aortic aneurysm to diagnose. The chief symptom is pain,

conducted to the vessels of the neck. The differential diagnosis is from aortic stenosis (presence of pulsation and retrosternal dullness, while the heart is not enlarged unless there is aortic valve involvement). The aneurysm may be confined to the ascending aorta, or may involve part of the arch as well, in

FIG 59B.—Left oblique view The fusiform aneurysm is well seen in the ascending aorta lying above the heart shadow The remainder of the aorta is widened and tortuous

which *case the signs and symptoms of aortic arch aneurysm* are present also. In advanced cases the aneurysm sometimes erodes through the chest wall (ribs or sternum), appearing as a pulsating swelling beneath the skin (Fig 60)

(b) *Arch of aorta* ("the aneurysm of symptoms") There is

nerve ; the cough then has a peculiar, hollow quality, and

gives rise to dysphagia as well as pain. The differential [illegible] of aneurysm of the descending thoracic aorta is from [illegible]

B

Fig 60.—Cardio-aortic syphilis (contd).

B *Photograph of swelling, profile view*

(d) *Abdominal aorta* Syphilis is a rare cause of aneurysm of the abdominal aorta, most cases are the result of atheroma. The aneurysm appears as a pulsating swelling in the middle line above the umbilicus The pulsation is expansile, a point which distinguishes aneurysm from tumours exhibiting transmitted pulsation The differential diagnosis is from carcinoma of stomach, carcinomatous pre-aortic glands, lymphadenomatous pre-aortic glands, tumours of pancreas, and from pulsation due to a normal aorta, aortic pulsation in a patient with lordosis easily simulates an aneurysm.

usually a continuous, dull boring pain in the back which is peculiarly persistent though it may become aggravated from time to time; in some cases it is made worse by effort. There is usually little to find to account for it—physical signs are

A

Fig 60 —Cardio-aorti[illegible] of ascending aorta eroding chest wall [illegible] of the sternum; [illegible] become larger, and has caused dull, aching pain, occa[illegible] pain Heart enlarged, aortic systolic and diastolic murmurs present, BP 146/46 Electrocardiogram shows inversion of T in all leads, axis is normal, but a deep S3 is present. Wassermann reaction + +

A *Photograph of swelling*, anterior view

as a rule wanting. Occasionally a systolic murmur is audible in the back just to the left of the spinous process. Unexplained persistent pain in the back should always raise the suspicion either of aneurysm, or of metastases in the spine, and should lead to X-ray examination. Occasionally an aneurysm here

however, that a small percentage of cases are sero-negative, and that patients who have recently been taking mercury may give a negative reaction. It should also be pointed out that the Wassermann reaction may be temporarily positive in some

B

FIG. 60.—Cardio aortic syphilis (*contd.*).

D

acute

preva-

TREATMENT

There are two aspects to the treatment of cardio-aortic syphilis, first, the treatment of the cardiac symptoms as such, and secondly, the treatment of the syphilis.

Diagnosis of Cardio-aortic Syphilis

The main points in the differential diagnosis have been enumerated in describing the symptoms and signs of the various clinical syndromes which syphilis may produce. It should be suspected when angina of effort or coronary occlusion

C

Fig 60.—Cardio aortic syphilis (*contd*).

C *Antero-posterior teleradiogram* The semicircular shadow of the aneurysm projects from the right border of the mediastinal shadow The aortic knob appears widened, and the prominent left ventricular contour suggests left ventricular hypertrophy

appears below the age of 40, when aortic valvular disease develops after the age of 20; when a ringing second aortic sound occurs in the absence of hypertension, when symptoms or signs of an intrathoracic tumour appear in a relatively young patient, or when these are accompanied by aortic valvular murmurs. and in patients complaining of persistent pain in the dorsal region of the back for which no obvious cause can be found.

The Wassermann reaction is positive in the majority of cases of cardio-aortic syphilis. It is necessary to remember,

CHAPTER 12

THE CARDIOVASCULAR SYSTEM IN OTHER INFECTIONS

INFECTIONS other than rheumatic fever and syphilis may affect the cardiovascular system, causing death from circulatory failure during the acute stage. With a few exceptions, however, the tendency is for complete recovery to occur if the acute stage is survived; these other infections are therefore less important as causes of chronic heart disease. In some circumstances there is actual infection of the heart, exemplified by septic or tuberculous pericarditis and by streptococcal, staphylococcal, pneumococcal, gonococcal, or meningococcal endocarditis, conditions which have already been described in Chapters 8 and 9. In other instances there is toxic necrosis of the heart muscle, exemplified by diphtheritic myocarditis, analogous necrosis occasionally occurs in other severe infections. More frequently, however, acute infections give rise to a primary peripheral circulatory failure, the result of toxic damage to the capillaries. The cardiovascular complications of some of the more important infections other than rheumatic fever and syphilis are considered in this chapter

1 THE HEART IN DIPHTHERIA

Circulatory failure is one of the most serious complications of diphtheria Two varieties have been described, peripheral and central, the early fall in blood pressure which may be associated with collapse is attributed to peripheral failure, while the later acute circulatory failure is thought to be myocardial It is possible, however, that both varieties may occur in the same case Pathologically, diphtheria causes a myocarditis characterised by infiltration of the muscle fibres with fatty globules In some cases there are areas of necrosis accompanied by perivascular leucocytic infiltration. Post mortem the heart is dilated, its walls soft and friable, and thrombi may be present in the ventricles or auricles.

Clinically, the first evidence of circulatory failure is often a fall in blood pressure, the systolic pressure is apt to be

The cardiac manifestations are treated in exactly the same way as they would be if the case were non-syphilitic. Where there is angina of effort or breathlessness on effort, the patient's activities are restricted accordingly—that is to say, as much as is necessary and no more. Cardiac failure, or severe anginal pain, is treated by complete rest in bed. Morphine is given for severe pain or distress, and digitalis is used for cardiac failure. Note that the cardiac treatment comes first—there is not much *point in curing the syphilis if the patient is allowed to die of* cardiac failure in the process.

It is important to recognise precisely what one is attempting to do when one treats the syphilis. The chief aim of antisyphilitic treatment should be the *prevention* of cardio-aortic complications by adequate treatment in the primary and early secondary stage. Once vascular complications have developed, antisyphilitic treatment cannot repair the damage; all it can hope to do is to prevent further damage. In a recent case, this should be attempted: a course of potassium iodide followed by mercury and bismuth should be given, but the organic arsenical preparations are contra-indicated; they are dangerous, especially in cases with coronary orifice stenosis, where they may cause sudden death. Alternatively penicillin may be used, the initial dose should be small because there is a risk of a Herxheimer reaction with penicillin as with organic arsenical compounds the dose may be gradually increased from 500 up to 50,000 units. If the Wassermann reaction remains persistently positive after two or three *adequate courses, there is no point in continuing*, the patient, not the Wassermann reaction, should be treated Again, in long-standing cases with gross cardiac and aortic damage, there is little to be gained by antisyphilitic treatment, cardiac treatment *is much more important*.

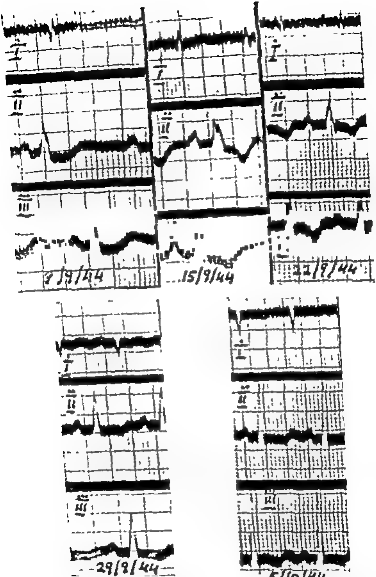

FIG 61—Serial electrocardiograms at weekly intervals from a case of diphtheritic myocarditis Male, aged 23 Gravis infection Onset on

the last electrocardiogram), and death occurred on the 40th day

affected to a greater extent than the diastolic. Pallor and collapse frequently follow or accompany the fall in blood pressure. Myocardial involvement is often ushered in by irregularity of the pulse; this may be due to extrasystoles, to partial block with dropped beats, or to auricular fibrillation; Neubauer noted paroxysmal tachycardia in 7 cases (supraventricular in 4, ventricular in 3). The first heart sound becomes soft, sometimes almost inaudible. The electrocardiogram may show distortion of the ventricular complexes (see Fig. 61), bundle-branch block, intraventricular block, or a disorder of rhythm The pulse is feeble and the blood pressure low. Ultimately acute myocardial failure develops, sometimes with considerable suddenness. There is pallor and vomiting, with epigastric or praecordial pain, breathlessness, and collapse; the pulse becomes extremely irregular, rapid, and feeble: rales appear at the pulmonary bases, the liver becomes enlarged, and acetone is often present in the breath. In some cases it is possible to demonstrate the cardiac enlargement clinically, but more often not. Death occurs in a high proportion of the patients who develop signs of symptoms of myocardial failure.

In those patients who have shown evidence of circulatory involvement and who survive, convalescence is often protracted, but complete recovery is the rule. It is sometimes stated that diphtheria can cause permanent cardiac damage Personally I have never seen a case, though I have had a number of patients referred who were alleged to be suffering from post-diphtheritic heart disease. These cases fell into two groups. In the largest group there was no evidence of any organic heart lesion; the patient's condition was an effort syndrome, most of them showed evidence of anxiety concerning their hearts and had clearly had a bad fright about it The patients in the second group showed an organic heart lesion; but in all of them it was a valvular lesion of rheumatic type, and there was a history of rheumatic fever, chorea, tonsillitis, etc, as well as of diphtheria. Leys has recently published a case of persistent heart block in a patient with a history of diphtheria but no rheumatic fever, etc , it is questionable whether the absence of a rheumatic history can be taken as proof that the lesion is not rheumatic, but if it is accepted as a genuine diphtheritic lesion, the case must be regarded as an exceptional one.

Treatment.—There is probably no condition in which the

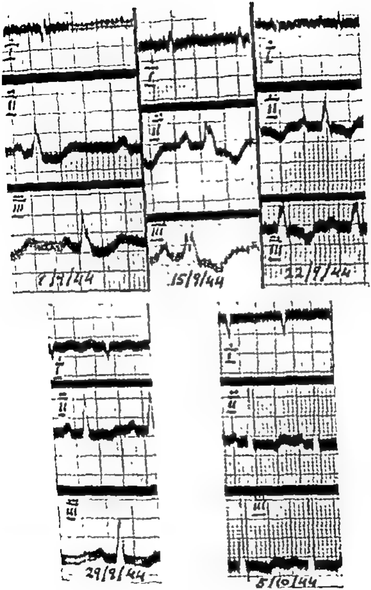

FIG 61 — Serial electrocardiograms at weekly intervals from a case of

statement that " prevention is better than cure " is more true. Once signs of myocardial failure appear in diphtheria, treatment is probably of little avail, the difference between death and survival being largely a matter of chance. The most obvious method of prevention is clearly immunisation. Failing immunisation, the best chance of preventing myocarditis lies in early diagnosis, early use of antitoxin in adequate dosage, and strict rest from the onset of the illness. The early fall in blood pressure due to peripheral circulatory failure calls for removal of pillows, elevation of the foot of the bed, and application of an abdominal binder. Nikethamide ("coramine"), "cardiazol", or *strychnine are possibly of value in this type*, *some authorities* recommend a combination of atropine sulphate (gr. $\frac{1}{100}$), strychnine sulphate (gr. $\frac{1}{100}$), and adrenaline (m. 5 of a 1 in 1000 solution) given four-hourly. In myocardial failure none of these drugs is of any avail, and drugs of the digitalis group are more likely to do harm than good. Glucose should be given by mouth, or if vomiting renders this route impracticable, intravenously. When there is ketosis, sodium bicarbonate should be given. By the time symptoms of cardiac failure appear, damage to the heart muscle has already been done, and it is too late to hope for any results from further antitoxin, though this is sometimes given.

In patients who have survived the acute stage of myocarditis, rest should be continued until the normal tone of the heart sounds has been restored, blood pressure has returned to normal, any irregularity of pulse has disappeared, and the electrocardiogram has returned to normal Thereafter convalescence should be slow, graduated, and carefully supervised. It is important, in the stage of convalescence, to guard against the development of an anxiety state, and to make it clear to the patient that there has been no permanent damage to the heart. Cases in which effort syndrome develops as a sequel to an attack of diphtheria should be treated along the usual lines for effort syndrome (Chapter 20)

2 Pneumonia

The fever in pneumonia (as in other acute infections) entails an increased metabolism and increased circulation rate At the same time consolidation offers obstruction to the passage of

blood through parts of the lung, thereby increasing the work of the right ventricle. Despite this, cardiac failure is extremely rare in persons whose hearts are healthy at the onset of a pneumonia; circulatory failure in them is almost always peripheral. When pneumonia supervenes in a patient with an already damaged heart it may precipitate cardiac failure; this is especially likely when the original lesion has caused right ventricular hypertrophy (mitral stenosis, chronic lung disease, congenital heart disease) or when left ventricular failure has been present for some time and has secondarily affected the right ventricle. The resulting failure is right ventricular with cyanosis, distension of the neck veins, hepatic enlargement, and often oedema.

The common type of circulatory failure in pneumonia is peripheral. The manifestations differ little from those of shock. There is pallor or pale cyanosis; the pulse rate rises and the blood pressure falls; the heart is not enlarged; the sounds are embryocardial; there is no venous distension, no hepatic enlargement and no oedema.

Left ventricular failure is extremely uncommon in pneumonia, except in persons in whom it has been present or imminent prior to the onset; in them the increase in the circulation rate may be sufficient to precipitate or aggravate failure. Pulmonary oedema in a case of pneumonia is not necessarily evidence of left ventricular failure; it can result from toxic damage to the lung capillaries.

Direct infection of the heart arises in pneumonia occasionally. Pericarditis is said to occur in about 2 per cent of cases; it may be fibrinous or purulent. Endocarditis is less frequent; it is an acute bacterial endocarditis and affects the aortic valve more often than the mitral.

Effort syndrome and neurocirculatory asthenia are rare after primary pneumococcal pneumonia, but frequent in convalescence from influenzal pneumonia.

Treatment.—As in the case of diphtheria, prevention of circulatory failure is easier than cure. The risk of failure, especially the peripheral type, can be considerably reduced by early use of sulphonamides or penicillin in adequate dosage, by sound nursing, and by ensuring sufficient sleep during the initial days of the illness. Many lives have been saved by the judicious use of morphine at the correct time, viz. when

pleuritic pain is associated with restlessness or insomnia during the first few days. Unnecessary moving of the patient should be avoided; *when removal to hospital is desirable it should* take place at the earliest possible moment, preferably not after the third day; the mortality increases with each day's delay Once signs of peripheral failure have appeared, the prognosis is bad, patients who reach the "slaty" colour rarely recover. I doubt whether drugs make any difference at this stage, though coramine, cardiazol, strychnine, and alcohol all have their devotees. Digitalis is harmful in cases of peripheral failure, it should be reserved for the less frequent cases of genuine cardiac failure. *The treatment of pericarditis and* endocarditis has already been discussed (Chapters 7 and 8), while effort syndrome will be dealt with in Chapter 20.

3. Influenza

When influenza is complicated by pneumonia the considerations in the preceding section apply. In uncomplicated cases there may be peripheral circulatory failure There is also some evidence that a true myocarditis may occur, thus dropped beats due to heart block or to sino-auricular block have been observed, myocarditis, however, is infinitely less common than in diphtheria In patients with pre-existing *mitral stenosis, influenza not infrequently precipitates auricular fibrillation* Finally, influenza seems to be particularly prone to give rise to effort syndrome during convalescence; this can sometimes be traced to an inadequate period of rest in bed after defervescence It can be taken as a sound general working rule that the period of rest in bed after defervescence in any acute illness should approximately equal the duration of the pyrexia; and convalescence should equal the total period of rest in bed

4. Typhoid Fever

Circulatory failure in typhoid fever is usually peripheral. It may be precipitated by the severity of the toxaemia, by the reflex shock of perforation, or by loss of fluid from haemorrhage The manifestations are the same as in other acute infections,

and have been described under pneumonia On the other hand, a genuine toxic myocarditis occasionally occurs; toxic necrosis of the myocardium may be found at autopsy, and in these cases there may be a true cardiac failure. Coronary occlusion has been recorded in rare instances, no doubt a sequel to the uncommon typhoid arteritis.

5. Tuberculosis

A mistaken diagnosis of heart disease is by no means uncommon in pulmonary tuberculosis, particularly when the auscultatory signs of the lung lesion are not obvious. In young subjects with active lesions, the pallor is not unlike that of rheumatic carditis, there is usually persistent elevation of the pulse rate and often some limitation in the capacity for effort, add the physical findings of a diffuse cardiac impulse and an innocent murmur (both of which are frequent in tuberculosis as in other infective illnesses), and it is easy to see how the mistake arises, the case receives a label of "myocarditis" or "rheumatic carditis" The foregoing symptoms and signs are frequent with erythema nodosum, and no doubt account for the fact that cases of tuberculous erythema nodosum are often mistakenly regarded as rheumatic

A second class of case in which diagnostic difficulties arise is with chronic fibroid lesions in older subjects Here breathlessness is a prominent symptom, tachycardia is often present, and there may be displacement of the cardiac impulse simulating enlargement These mistakes can be avoided by refusing to accept tachycardia with a systolic murmur as evidence of heart disease, and by insisting on X-ray examination in such cases. In the chronic fibroid type, clubbing of the fingers may be present as a pointer to the diagnosis The reverse mistake of regarding rheumatic carditis as tuberculosis is also possible, I saw one young woman with mitral stenosis and congestive failure who had been discharged from a sanatorium a month or two previously, having spent several months there with alleged pulmonary tuberculosis, she had a series of X-ray reports describing "an active tuberculous lesion at the right apex", but the sputum had never been positive, a month later no trace of tuberculosis, either recent or old could be found at autopsy.

Tuberculous pericarditis. Pericarditis is the most frequent cardiac complication of tuberculosis. Most cases arise in the early stage of dissemination, usually by direct extension from a tuberculous mediastinal gland; it may consequently be found with a primary Ghon focus in the lung, with an apparently primary tuberculous pleurisy, or without obvious lung or pleural lesions Less often, pericarditis arises at a later stage, by extension from pleura or glands in cases with long-standing pulmonary lesions. Finally, it is sometimes a blood infection, in which case it is an incidental finding in the course of miliary tuberculosis and it does not modify the illness In its fully fledged form it passes through four stages. (1) an acute fibrinous pericarditis; (2) stage of effusion; (3) a stage of apparent recovery, and (4) a stage of pericardial thickening and constriction. The acute stages are often overlooked as the constitutional symptoms are usually mild, sometimes insufficient to lead the patient to seek medical advice, fever is slight or absent, tachycardia insignificant in comparison to that of rheumatic pericarditis, appetite remains good, and there is often a definite euphoria, a pressure cough is sometimes the sole complaint. In the effusive stage, symptoms are also mild unless tamponade develops Many cases are recognised for the first time in the final stage of constrictive pericarditis. There is evidence that the disease sometimes becomes arrested either before development of an effusion or after its absorption. On the other hand, a tuberculous effusion sometimes becomes loculated and chronic, leading to chronic tamponade. When tubercle bacilli are present in the pericardial fluid, the prognosis is bad, mortality being over 80 per cent, but when the fluid is sterile mortality is very low and the majority of patients make an apparent, though sometimes only temporary, recovery. Differentiation from rheumatic pericarditis is suggested by mildness of constitutional symptoms and absence of endocardial lesions, diagnosis is confirmed by demonstration of a pulmonary or mediastinal gland tuberculosis, by examination of the pericardial fluid, or by appropriate skin tests Aspiration and introduction of air into the pericardium has been recommended as treatment in the effusive stage, but many cases with a sterile effusion do well with conservative treatment. Streptomycin may be used to control the infection In the constrictive stage, the pericardium should be excised.

Tuberculous myocarditis. The frequency of tuberculous myocarditis in cases with pericarditis has varied from 3 per cent to 30 per cent in different series In the absence of pericarditis, myocarditis is rare There is one uncommon tuber- [illegible]

[illegible] it may be recognised on cardiographic examination, or may remain unsuspected; at autopsy, extensive tuberculous infiltration of the myocardium is found

Tuberculous endocarditis, if it occurs at all, must be extremely rare, most writers deny its existence. It is also rare to find rheumatic endocardial lesions in the presence of active tuberculosis, development of mitral stenosis in a tuberculous subject favours healing of the pulmonary lesion.

Peripheral circulatory failure is, on the whole, uncommon during the acute febrile episodes of tuberculosis, but it frequently accompanies the onset of a pneumothorax, and it may occur in association with a haemoptysis. Chronic fibroid phthisis is capable of producing cor pulmonale, the features of which are described in Chapter 16.

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intima while the underlying media is infiltrated by leucocytes; small aneurysms sometimes result (mycotic aneurysms), and these have been known to rupture or to form the starting-point of a dissecting aneurysm

Acute arteritis is followed by thrombosis of the affected vessel, and, in the case of an internal vessel, the symptoms are those of thrombosis followed by infarction in the organ concerned In a superficial vessel there is severe pain with redness, swelling, and tenderness along the course of the vessel, while the pulse disappears from the distal part of the vessel. The limb becomes pale and cold, and later cyanotic. As in the case of embolism (p 37), gangrene may result; alternatively improvement and return of the circulation to normal follows the development of adequate anastomotic channels. The attack is associated with fever.

Treatment consists of rest with measures for the relief of pain Local application of heat may give some relief, but morphine is generally required The use of papaverine to abolish

Chronic Arteritis

Chronic arteritis is, in most cases, due to syphilis. The nature and effects of the condition as met with in the aorta have been fully described in Chapter 11 Syphilitic endarteritis, in addition, affects the cerebral vessels, giving rise to cerebral softening, and it also occurs in association with syphilitic meningitis, the description of these conditions is properly a matter for a text-book on ne
of the present volu
local endarteritis i
but these lesions ... importance only and have no bearing on the general circulation

Periarteritis Nodosa

Periarteritis nodosa is an uncommon disease in which localised inflammatory swellings appear along the course of the medium-sized arteries, they are accompanied by a general febrile reaction The cause is unknown, the disease occurs in young adults, more often males than females.

CHAPTER 13

ARTERIAL DISEASE

Classification and General Considerations.—In the past there has been much confusion regarding the chronic degenerative forms of arterial disease. This has arisen partly because the different varieties of arterial degeneration may exist side by side and simultaneously in a single patient, and partly because the word "arteriosclerosis" has been used in different senses by different authors. Some use the term to cover all forms of chronic arterial degeneration (e.g Taylor's *Practice of Medicine*, Conybeare's *Textbook of Medicine*). A second group exclude atheroma as a distinct entity, and employ the word arteriosclerosis to cover the remaining forms of arterial degeneration, viz. diffuse hyperplastic sclerosis and hypertension, this is the practice followed by the majority of pathologists and many physicians. Yet a third group exclude cases of hypertensive disease as well as atheroma, using the term "Arteriosclerosis" only for cases of "diffuse hyperplastic sclerosis without high blood pressure". *I shall attempt, as far as* possible, to avoid the use of this controversial word in the present volume, but in case it should inadvertently creep in, it may be accepted that it is being used in its third and most restricted sense

Infective or inflammatory disease of the arteries will be described first, followed by the three main forms of **chronic degenerative arterial disease.**

1. Inflammatory Diseases of the Arteries

Acute Arteritis

This is a rare condition sometimes seen as a sequel to acute infections. Acute multiple arteritis is most often encountered as a sequel to typhoid fever, but has been described in other infections. Coronary arteritis has been described in rheumatic fever. Localised arteritis may develop in the aorta in cases of acute bacterial endocarditis, vegetations appearing on the

progress is very variable, it was formerly thought to be fatal within a few weeks to a few months; but some cases are now known to run an intermittent course for years.

TREATMENT.—No specific treatment is known to influence the course of the disease. Symptoms should be treated on general lines as they arise

Thrombo-angiitis Obliterans (Bürger's Disease)

In this condition there is inflammation of the deep arteries and veins, giving rise to thrombosis; in some cases there is also a superficial migratory thrombophlebitis ("thrombophlebitis migrans"). The cause is unknown The disease occurs predominantly in middle-aged males, and is said to be more common among Jews; but it is by no means confined to that race Excessive smoking, particularly of heavy pipe tobacco, has been blamed as a predisposing cause; alternatively, the condition has been attributed to infection, though the nature of the infecting agent is quite unknown.

PATHOLOGY.—There is progressive thrombosis in the deep vessels of the limbs, especially the legs; the walls of the vessels show only slight inflammatory changes, but they become vascularised and the thrombi become organised as the disease progresses Gangrene follows in due course, but the rate of progress varies greatly from case to case.

SYMPTOMS.—It has been customary to teach that the disease runs a progressive course characterised by pain on effort in the early stage followed by pain at rest later, and ultimately by gangrene Recent workers have shown that this is true only in one group of cases in which there is widespread involvement of the whole arterial tree of the limb. In the remaining cases the symptoms are determined by the level of the arterial block rather than by the stage of the disease.

Blockage of a main vessel gives rise to *intermittent claudication* as the presenting symptom, in these cases the external iliac, femoral, or popliteal artery is involved. Most often the patient complains that, after walking a certain distance, he develops pain or a painful cramp, in the muscles of one calf, after resting for a minute or two the pain eases and he is able to continue walking, but again only for a certain distance Pulsation is diminished or abolished in the dorsalis pedis and

PATHOLOGY.—The essential feature is a localised inflammatory lesion of the vessel, visible to the naked eye as a white or yellowish nodule; the artery is usually dilated to form a small aneurysm at the affected level. The inflammatory reaction involves all three coats of the vessel; these are infiltrated with polymorphonuclear leucocytes, lymphocytes, plasma cells, and in many cases eosinophil cells; there are areas of hyalinisation and necrosis Thrombosis occurs within the vessel at the seat of the lesion, and in the course of time the thrombus becomes organised, later canalised. In consequence of the thrombosis, infarction occurs in the organ supplied. The lesions are multiple, developing either successively or in crops; while any vessel may be affected, those of the heart, kidneys, and intestines are most often involved.

SYMPTOMS.—The illness is associated with irregular fever, local symptoms due to infarction, and in the later stages development of subcutaneous nodules, biopsy of which will confirm the diagnosis The onset is very variable. Fever and tachycardia with general weakness may precede any local symptoms. Alternatively, the first event may be sudden onset of pain due to infarction in one or other organ When the coronary vessels are involved, the illness may either simulate rheumatic carditis, or start abruptly with symptoms of coronary thrombosis. Lesions of the renal vessels give rise to an illness like nephritis with fever, albuminuria, haematuria, and casts in the urine From disease of the mesenteric vessels there may be acute attacks of abdominal pain; a picture resembling intestinal obstruction with pain, vomiting, and absolute constipation results from mesenteric thrombosis; perforation and peritonitis have been observed as sequelae Attacks of biliary colic have been described. Sometimes lesions occur in the pulmonary vessels where they can be responsible for haemoptysis, asthmatic attacks, or symptoms of bronchitis The fever is irregular, and there may be afebrile periods Blood examination shows no diagnostic features, there is a moderate anaemia and leucocytosis, cultures are sterile; and the Wassermann reaction is negative. The development of subcutaneous nodules in relation to a superficial artery is suggestive of the diagnosis, and biopsy provides the only means of certain diagnosis during life. Many cases are diagnosed only at autopsy. The general course of the disease is downhill, but the rate of

progress is very variable; it was formerly thought to be fatal within a few weeks to a few months; but some cases are now known to run an intermittent course for years.

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Blockage of a main vessel gives rise to *intermittent claudication* as the presenting symptom, in these cases the external iliac, femoral, or popliteal artery is involved Most often the patient complains that, after walking a certain distance, he develops pain or a painful cramp, in the muscles of one calf; after resting for a minute or two the pain eases and he is able to continue walking, but again only for a certain distance Pulsation is diminished or abolished in the dorsalis pedis and

malleolar arteries. In a second (and rather smaller) group the painful cramp affects the small muscles of the foot, behaving in precisely the same manner in relation to walking; blockage of the posterior tibial artery is involved in these cases, and pulsation behind the internal malleolus is abolished. In the absence of simultaneous blockage of the small vessels of the foot there is no pain at rest, no discoloration and no gangrene; the collateral circulation is sufficient to maintain the nutrition of the foot. Rarely the painful cramp affects the gluteal muscles, presumably from blockage of the gluteal branches of the internal iliac; I have encountered three such cases.

Obstruction of the smaller arteries of the foot such as the plantar arch or its digital branches produces pain in the foot which is felt at rest. The pain may be aggravated when the limb is allowed to hang down or when the patient stands for long; it is often worst at night. On allowing the limb to hang down from the edge of the bed, the toes become intensely flushed and red, or in more advanced cases livid and cyanotic: on elevating the foot above the level of the heart the toes blanch. At a later stage the toes are permanently discoloured, trophic skin changes soon develop, a perforating ulcer may form, and gangrene follows in a short time. The block being distal to the ankle, pulsation in the dorsalis pedis and malleolar arteries may be normal.

In about a quarter of the cases a superficial thrombophlebitis migrans occurs during the earlier stages of the illness. The thrombi are small and localised; the affected segment of the vein is acutely tender. The lesions undergo resolution, but fresh ones appear in some other vein, often in another limb.

Diagnosis.—The pain of intermittent claudication is sometimes mistaken for sciatica when it affects the gluteal or calf muscles, or for foot strain when it is felt in the small muscles of the feet; careful attention to its behaviour in relation to walking should prevent this mistake. When intermittent claudication is the presenting symptom, the diagnosis is from sclerosis of the media; this occurs at a rather later age, often above 50, while thrombo-angiitis has its maximum incidence between 35 and 50. In sclerosis of the media, X-ray examination often shows calcification in the affected vessels and general examination of the cardiovascular system will usually reveal evidence of the disease elsewhere. When thrombo-

phlebitis migrans is the presenting symptom, it should be borne in mind that this may be a symptom of malignant disease, of thrombo-angiitis obliterans, or may be the sequel to an infection. Pains in the feet and toes with redness of the dependent limb might suggest erythromelalgia, but in the latter the feet do not blanch on elevation and the arteries pulsate forcibly, gangrene does not occur.

TREATMENT.—Various forms of treatment have been recommended with claims that they will delay or even temporarily arrest the progress of the disease; but, so far, no specific treatment which will arrest it permanently is known. Bürger recommended "passive postural exercises"; with the patient lying in bed, the limb is first elevated and supported at an angle of from 60° to 90° until blanching occurs; it is then allowed to hang down over the edge of the bed until a good red colour has been present for one minute, then it is placed horizontal for 3 to 5 minutes and kept warm with a hot-water bottle. The total cycle occupies from 6 to 10 minutes, and these cycles are repeated over a period of an hour. I have seen marked temporary improvement follow this treatment, but the patient relapsed after a few weeks and despite continued treatment he developed gangrene.

"Intermittent venous occlusion" has also been advocated and has been stated to improve the circulation in the foot, to ward off gangrene, and to produce considerable relief of pain. A sphygmomanometer armlet is applied round the thigh and is connected to an electrically operated pump which alternately inflates and deflates the armlet to any desired pressure; the pressure chosen should be about 40 to 50 mm., i.e. sufficient to occlude the veins but not the arteries, and the speed of the apparatus is adjusted so that pressure is applied for two minutes and then released for two. It is possible to set up an apparatus operated by water on the siphonage system, and I have seen such an apparatus effectively used; but it is difficult to adjust and it may not operate constantly if the pressure in the water mains varies. With either apparatus, treatment may be given for an hour night and morning to begin with, and, if its effects are well tolerated, its duration may be prolonged to several hours daily.

In suitably selected cases lumbar sympathectomy gives relief for periods up to a year or two; but the disease is apt

to be progressive and symptoms tend to recur in due course. The results are good in cases of blockage of the external iliac, femoral, or posterior tibial arteries, and in cases with isolated blockage of the small arteries of the foot. They are poor when the popliteal artery is obstructed as the branches to the gastrocnemius are usually implicated and the collateral circulation is poor. When obstruction is widespread, giving rise on the one hand to intermittent claudication with obliteration of the pulse in the main vessels, and on the other hand to pain at rest with discoloration of the toes, sympathectomy is useless, amputation above the knee is inevitable. Sympathectomy may be preceded by trial of the effects of local blocking of the sympathetic trunks by procaine; if this temporarily relieves the symptoms, a successful result from sympathectomy may be anticipated; nevertheless a poor response does not necessarily contra-indicate the operation, as the development of a collateral circulation is a gradual process. In doubtful cases, arteriography may give valuable information.

Once gangrene has appeared, amputation becomes necessary; the level depends on the site of the arterial obstruction. When the proximal vessels are free, local amputation may suffice, especially if a sympathectomy has been performed; should gangrene reappear in the stump, amputation below the knee will be required. When the proximal vessels are blocked, amputation must be above the knee

Drugs are of little value. Iodides and trinitrine have been used. Gentle massage, heat, diathermy, and ultra-violet light have also been used. Their effect may be soothing, but they make little difference to the outcome of the disease For severe pain or cramp occurring at rest, morphine or papaverine may be necessary if other methods fail to give sufficient relief to permit sleep; needless to say, the use of morphine in a slowly progressive condition of this nature is undesirable if it can be avoided by any other method of treatment.

Temporal Arteritis

This is an uncommon inflammatory disease of the arteries which occurs in elderly people, more often women than men The cause is unknown but the pathology and the presence of fever suggest an infective process.

PATHOLOGY.—A subacute inflammation starts in the adventitia and spreads by the vasa vasorum to the media. Focal areas of necrosis result from interference with the blood supply; cellular infiltration follows and ultimately the media is largely converted into chronic granulomatous tissue with lymphocytes, plasma cells, and monocytes, giant cells are found here and there The internal elastic lamina undergoes necrosis in places, proliferation in others, giving rise to loops which may divide the granulation tissue into follicles resembling tubercles. In the smaller arteries thrombosis usually follows and the thrombus rapidly becomes organised

SYMPTOMS.—General symptoms usually precede the arterial thrombosis, sometimes for several months. There may be muscular aches, pains in the knees, shoulders, and hips, fever, night sweats, weakness, anorexia, and marked loss of weight. Headache is constant, beginning as a generalised ache, later becoming severe and localised, it may be frontal and temporal, or occipital and cervical depending on whether the temporal or occipital arteries are affected

The diagnostic feature is acute inflammation, most often in the temporal artery but sometimes in the occipital or in a cerebral vessel, rarely a limb artery is involved The affected artery is red, tender, and swollen, with the onset of thrombosis pulsation disappears it may return when the inflammation subsides The cervical glands are usually enlarged. Rarely the skin breaks down The blood pressure in many cases is normal, high readings are probably due to pre-existing hypertension. Moderate anaemia and slight leucocytosis are usually present, there is no eosinophilia Involvement of a cerebral artery produces a picture suggesting isolated or multiple cerebral thrombosis The retinal vessels have been affected in some cases with corresponding visual symptoms

DIAGNOSIS.—The early ...
cerebral thrombosis trigem...
sitis, neuritis, or osteoarthri... The clinical course and ultimate temporal artery thrombosis make the diagnosis clear

PROGNOSIS.—The majority of patients ultimately recover after 6 to 12 months A few have died ... e remain

...noted im-
temporal

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Temporal Arteritis

This is an uncommon inflammatory disease of the arteries which occurs in elderly people, more often women than men The cause is unknown but the pathology and the presence of fever suggest an infective process

lesion acquires a chalky consistency and a chalky white colour. The surface of the intima frequently ulcerates (atheromatous ulcer) and thrombus forms on the base of the ulcer, giving it a dirty reddish-brown or chocolate colour. Meantime the media overlying the atheromatous ulcer becomes the seat of a secondary fibrosis. An atheromatous ulcer may form the starting-point for a dissecting aneurysm, and it may so lead indirectly to rupture of the artery. The vessels chiefly involved are the large and medium-sized arteries. The aorta is very commonly affected, especially the abdominal aorta where the most advanced changes are often seen. The coronary and the cerebral vessels are other frequent and important sites. The mesenteric vessels, renal vessels, and other arteries of like size may also be affected. In some cases a patch of atheromatous infiltration is seen at the base of the mitral valve cusps, but in this situation it does little harm unless it progresses to calcification.

Effects of Atheroma.—The effects of atheroma are local, and are attributable to ulceration which provides a basis for thrombus formation. Thus atheroma is the most common cause of arterial thrombosis. The cerebral arteries and the coronary arteries are the most frequent sites though other vessels such as the mesenteric or the limb arteries may be affected. Total occlusion of a vessel such as a coronary artery causes a myocardial infarct, incomplete occlusion leads to ischaemic fibrosis, these conditions are discussed in the section dealing with coronary disease (p. 318). The cerebral vessels are thin-walled and inadequately supported, atheromatous ulceration here may permit of rupture in response to any sudden rise in blood pressure such as might be caused by sudden effort or strain. The *development of* a dissecting aneurysm of the aorta with secondary rupture of the aorta has already been mentioned. In the abdominal aorta ath[illegible]

Atheroma by itself has no effect whatever on the blood pressure. In cases where *extensive* atheromatous ulceration has led to widespread secondary fibrosis in the media, an effect similar to that of primary fibrosis of the media is produced. The *only* effects of atheroma on the heart are those

artery which had been carried out for diagnostic purposes; he recommends this as a therapeutic measure. He also reports benefit from nicotinic acid in doses of 300 mg. daily. Potassium iodide and thiamine have been recommended by other authors.

2. Chronic Degenerative Arterial Disease

There are three forms of chronic degenerative arterial disease. These are:

(1) *Atheroma* (atherosclerosis), primarily a disease of the intima.

(2) *Medial sclerosis* (synonyms: "arteriosclerosis", medial fibrosis, medial calcification, senile arterial sclerosis, Monckeberg's sclerosis, diffuse hyperplastic sclerosis without high blood pressure), primarily a disease of the media of the large and medium-sized vessels.

(3) *Hypertension* (hypertensive disease, diffuse hyperplastic sclerosis with high blood pressure, "arteriolosclerosis"), primarily a disease of the arterioles.

Any one of these conditions may occur alone, without either of the others Each is a disease of the second half of life yet differs slightly from the others in its maximum age incidence. Each has its own characteristic effects on the heart, the blood pressure, and the kidneys, and each produces its own symptomatology in its own particular way They are all very common conditions, and they are not mutually exclusive, consequently it frequently happens that two of them, or all three, occur simultaneously in a particular patient An additional reason for confusion lies in the fact that atheromatous ulceration of the intima leads to secondary sclerotic changes in the overlying media, while fibrosis of the media leads to secondary endarteritis.

Atheroma or Atherosclerosis

The primary change here is a lipoid degeneration of the cells of the intima Lipoid droplets appear in groups of adjacent cells, giving rise to irregular yellow streaks or plaques. These may be quite flat or slightly raised; their colour and shape distinguish them from syphilitic plaques In the course of time the cholesterol esters become replaced by calcium salts, the

cutaneous tissues giving rise to extensive ecchymoses. Rupture into the pericardium with development of a haemopericardium and death from cardiac tamponade has been recorded; in other instances rupture has been into the pleural or peritoneal cavity. Even in the absence of a dissecting aneurysm, an atheromatous aorta may rupture in consequence of a blow or of a sudden severe strain; death usually ensues within a matter of minutes.

AETIOLOGY.—The causation of atheroma is still a matter of controversy. An early stage in atheroma is not infrequently seen in persons dying during adolescence ("atheroma of puberty"—Aschoff), but seems to be reversible as atheroma is less often seen in those who die in early adult life. It becomes frequent again once the age of 40 has been passed, and its frequency increases steadily thereafter with advancing age. There are striking variations in its geographical incidence. In Northern Germany (Berlin) it is almost universal after middle life, and often extreme, in Southern Germany (Baden and Black Forest district) it is altogether much less frequent and on the average much less severe. The German school tend to attribute atheroma to dietetic factors, and have shown that mild grades of atheromatous change can be produced by feeding rabbits on a high cholesterol diet. The French school, on the other hand, tend to attribute atheroma to repeated *infections*; they point out that the response to infection commonly includes changes in the metabolism of lipoids and alterations in the blood cholesterol content. Yet other views have been that atheroma is a result of constantly recurring mechanical stress, the common distribution of atheromatous lesions in the aorta [illegible] present.

[illegible] is purely symptomatic.

Sclerosis of the Media

This disease, the many synonyms for which have already been enumerated, is *primarily a disease of the media* in which muscle and elastic tissue become replaced by fibrous tissue, at a later stage calcium salts may be deposited in the media, and in extreme cases the whole vessel is converted into a calcareous tube (Fig. 62). The arteries become elongated, and

resulting from coronary thrombosis (myocardial infarction) or partial coronary occlusion (para-arterial fibrosis); as long as atheroma remains limited to vessels other than the coronaries it has no effect on the heart and does not cause left ventricular *hypertrophy. The kidneys are unaltered.*

SYMPTOMS.—The clinical syndromes therefore which may be associated with atheroma include: coronary thrombosis, chronic coronary sclerosis, dissecting aneurysm of the aorta, rupture of the aorta, aneurysm of the abdominal aorta, *cerebral thrombosis, cerebral haemorrhage, mesenteric thrombosis,* thrombosis of other arteries (less common).

With the exception of dissecting aneurysm and rupture of the aorta, these syndromes are described elsewhere in this volume. A patient with a *dissecting aneurysm* often experiences *two painful episodes separated by an interval. The first attack* of pain represents the initial rupture through an atheromatous ulcer into the tunica media; the second is associated with the final rupture to the exterior or back into the lumen of the vessel. If the initial rupture occurs in the ascending aorta there is sudden severe *pain in the front of the chest, sometimes* with radiation to the arms, and accompanied by shock, clinically the attack resembles a coronary occlusion, transient cardiographic signs of *myocardial ischaemia* occurred in one of my cases, but the cardiographic signs of myocardial infarction are absent. With initial rupture occurring in the descending thoracic aorta the pain is felt in the back, but is still associated with shock. Extension of the aneurysm along the tunica media may or may not be *accompanied by further attacks of pain.* Irregular enlargement of the aorta is sometimes recognisable radiologically, and it may produce pressure symptoms pointing to aneurysm of the affected part. When the point of entry is proximal to the innominate or left subclavian artery the corresponding *pulses are abolished or greatly reduced*

Rupture of a dissecting aneurysm commonly takes place to the exterior; but occasionally the aneurysm ruptures back into the original lumen of the aorta, in which case the re-entrant rupture is not necessarily fatal. Rupture is associated with a second *attack* of severe pain and shock, the site of the pain depends on the level of the rupture. Death may follow in a few minutes; but sometimes patients survive for a day or two, in which case the *effused blood finds* its way into the sub-

may lead to complaint of pulsation or throbbing in the neck or head, and this is apt to be worse during exertion. The arteries are less responsive to vasoconstrictor and vasodilator influences, and consequently the circulation is less readily adjusted to changes in posture or bodily activity, with resulting faintness and dizziness These symptoms are especially apt to be felt if the patient rises quickly after stooping or reclining, or if he makes some sudden effort from resting, his inability to constrict his mesenteric vessels in order to compensate for the effect of gravity produces temporary cerebral anaemia. Local symptoms may arise in the same way. Thus the coronary vessels should normally dilate when an increased cardiac output is required; sclerotic vessels are incapable of dilating, with the result that the blood supply to the myocardium, adequate while the patient is at rest, becomes inadequate during effort, and myocardial pain develops (angina of effort). A similar failure on the part of the vessels of the legs to dilate in response to muscular exercise gives rise to "intermittent claudication"; this is a severe pain in the muscles of the leg which develops as soon as the patient has walked a certain distance; it forces him to stop; and it passes off quickly once he has stopped (Fig. 62). (Other causes of intermittent claudication include thrombo-angiitis obliterans and arterial thrombosis.) Rigid vessels are more liable to rupture in response to a sudden rise in blood pressure, hence haemorrhages may occur, and they may be either internal or external (cerebral haemorrhage

[illegible] it is usual to attribute it to the process of ageing, and to general wear and tear. It certainly becomes more frequent in each decade. It seems to be about 10 to 15 years later than hypertension in its main incidence In some patients it occurs in pure form without either atheroma or hypertension, in others it develops as a late sequel to one or other of these conditions. I have seen extensive arterial calcification in a woman aged 44 who had malignant hypertension as well as extensive atheroma, but calcification to this extent is exceptional in these circumstances and at this age. I have also seen widespread calcification develop in a young man who was treated with large (one might well say "heroic") doses of parathormone and vitamin D for tetany; but it is

Unless there is concomitant atheroma or secondary endarteritis, the heart muscle remains healthy, but should either of these conditions be present, the heart will be the seat of para-arterial fibrosis. The kidney, in a case of pure medial sclerosis, is of

Fig 64 —Sclerosis of the media

Showing tortuous "unfolded" aorta The ascending aorta projects further to the right and the descending aorta further to the left owing to widening of the curve of the arch Female, aged 46, complaining of menopausal symptoms BP 102/104 Cardiogram normal

normal size; it may show a few isolated deep scars on its surface (the "senile arteriosclerotic kidney"), clinically there may be occasional slight albuminuria, but renal function remains sound.

Symptoms.—The clinical symptoms are referable to rigidity and loss of elasticity in the vessels. The increased pulse pressure

may lead to complaint of pulsation or throbbing in the neck or head, and this is apt to be worse during exertion. The arteries are less responsive to vasoconstrictor and vasodilator influences, and consequently the circulation is less readily adjusted to changes in posture or bodily activity, with resulting faintness and dizziness. These symptoms are especially apt to be felt if the patient rises quickly after stooping or reclining, or if he makes some sudden effort from resting; his inability to constrict his mesenteric vessels in order to compensate for the effect of gravity produces temporary cerebral anaemia. Local symptoms may arise in the same way. Thus the coronary vessels should normally dilate when an increased cardiac output is required; sclerotic vessels are incapable of dilating, with the result that the blood supply to the myocardium, adequate while the patient is at rest, becomes inadequate during effort, and myocardial pain develops (angina of effort). A similar failure on the part of the vessels of the legs to dilate in response to muscular exercise gives rise to "intermittent claudication"; this is a severe pain in the muscles of the leg which develops as soon as the patient has walked a certain distance; it forces him to stop; and it passes off quickly once he has stopped (Fig. 62). (Other causes of intermittent claudication include *thrombo-angiitis obliterans* and arterial thrombosis.) Rigid vessels are more liable to rupture in response to a sudden rise in blood pressure; hence haemorrhages may occur, and they may be either internal or external (cerebral haemorrhage,

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questionable whether this is aetiologically related to the natural disease found in elderly persons.

Treatment.—No known treatment *will* influence the condition of the arteries—it is now universally accepted that the reputation formerly enjoyed by potassium iodide was undeserved—and treatment of the resulting symptoms is purely symptomatic.

Hypertension

Hypertension is primarily a disease of the arterioles (arteriolosclerosis), which become the seat of lipoid degeneration and *probably* spasm *The condition is generalised, with special* incidence on the arterioles of the kidney and retina. It gives rise to an increase in the peripheral resistance with a consequent rise in the diastolic pressure and a corresponding rise in the systolic pressure. *As a result of the raised blood pressure*, hypertrophic changes occur in the arteries and heart.

Aetiology.—Hypertension may be secondary to some other disease, but it often occurs as an apparently primary condition.

Secondary hypertension develops in some cases of chronic kidney disease, in lead poisoning, in gout, and in certain endocrine disorders.

(1) *Kidney disease* Hypertension occurs in those forms of kidney disease in which more than 75 per cent of the total glomeruli are either destroyed or temporarily out of action. Thus transient hypertension occurs in acute nephritis Hypertension is frequent though not invariable in chronic glomerulonephritis ("secondary contracted kidney"), in renal fibrosis secondary to obstruction whether due to prostatic enlargement or to urethral stricture, and in polycystic disease. In lead poisoning and gout, it is probably the result of toxic damage to the kidneys. Occasionally hypertension follows unilateral kidney disease such as pyelonephritis, and in these circumstances it is apt to run a rapid and progressive course (malignant hypertension); yet the majority of individuals with unilateral kidney disease retain a normal blood pressure

(2) *Endocrine disorders.* Hypertension is characteristic of suprarenal medullary tumours *(phaeochromocytomata)*; in the early stages it is usually paroxysmal, later becoming permanent; the syndrome is described in Chapter 17, p 376 Hypertension occurs as an integral part of the Pituitary

Basophil syndrome (Cushing's syndrome), and is also found in some cases of acromegaly. It is so frequent in elderly diabetics that it seems likely that the two conditions are causally related It is also relatively common in myxoedema. It occurs occasionally in thyrotoxicosis, especially the pituitary type of thyrotoxicosis, but its incidence in thyrotoxicosis does not seem to be any greater than, if as great as, in the general population at the same age. (I would emphasise that these remarks refer to genuine hypertension with persistently raised diastolic pressure, and not to the raised systolic with normal or low diastolic pressure found so characteristically in thyrotoxicosis.) Finally, hypertension in many women develops at the time of the menopause.

Primary hypertension or "Essential Hypertension" is the term used when hypertension develops in the absence of any of the causes mentioned. It is extremely common, accounting for something like 25 per cent of all cases seen in cardiological practice Its greatest incidence is in the age group 45-55 and *it affects the sexes* in about equal proportions, but it may develop at a much earlier age, sometimes even in the late teens A family history is frequently obtained, especially in the case of younger patients In younger patients the disease is apt to run a particularly rapid course (malignant hypertension). The cause of primary hypertension, whether benign or malignant, is unknown, but various theories have been suggested

(a) That the primary change is in the arterioles of kidney, due to some unknown toxin, and that this leads to kidney damage and so to hypertension Partial occlusion of a renal artery causes *hypertension* in experimental animals, it has been suggested that the partially anoxaemic kidney secretes renin which reacts with a pre-existing component of blood (hypertensinogen) to form a pressor substance (hypertensin). Hypertension so caused will *itself* lead to changes resembling those of malignant hypertension in the opposite kidney ...

... they certainly do not prove that all cases of primary hypertension are caused ...

... was constricted at its origin by

a ring of scar tissue resulting from atheroma; the right kidney was much atrophied while the left was of normal size but showed the changes of malignant hypertension. Though I have witnessed numerous autopsies in cases of primary hypertension, this is the only instance in which any such clue to the aetiology has been found.

(b) That the condition is of endocrine origin—by analogy. There is no proof other than analogy to support this view.

(c) That the rise in blood pressure is the primary event and is caused by vasomotor activity (Allbutt's view); on this view, the changes in the arterioles are the result, not the cause, of the raised pressure. The changes which appear in the opposite kidney in animals with experimental hypertension support this theory. The rise in blood pressure may be due to a life of stress, either physical or mental, to excesses in food or drink, or to emotional disturbances. Some believe that an anxiety state can lead to hypertension, which they regard as a "psychosomatic disease" This view is unproved, there is some evidence to support it, notably the facts that in one group of hypertensive patients symptoms of nervous instability akin to those of an anxiety state are prominent, and that certain cases are alleged to have been cured by psychotherapy On the other hand, many hypertensive patients exhibit none of the features of anxiety, being placid and emotionally stable, while many cases of anxiety neurosis continue for years without developing hypertension.

The truth regarding the aetiology of benign and malignant hypertension is probably to be found in the view that multiple factors are capable of causing it Hypertension should be regarded as a symptom-complex which may arise in various ways but which leads to secondary structural changes

PATHOLOGY —The earliest change is a lipoid degeneration in the walls of the arterioles, especially in kidney and retina, but also elsewhere This is followed by hypertrophy of the arterioles, possibly induced by spasm. As a result of the raised blood pressure, hypertrophy next affects the media of the arteries, especially the aorta, the muscular layers hypertrophy while the elastic lamina splits into several layers Concentric hypertrophy of the left ventricle appears simultaneously. At a later stage the hypertrophy of the media may be followed by sclerosis and deposition of fibrous tissue (diffuse hyperplastic sclerosis).

In consequence of the changes in the afferent arterioles of the glomeruli in the kidneys, the glomeruli undergo hyaline degeneration, and the corresponding tubule atrophies; its place is filled by fibrous tissue overgrowth (replacement fibrosis). The kidneys become reduced in size with a granular surface; the capsule strips fairly easily; the colour remains red (primary contracted kidney, arteriolosclerotic kidney, small red kidney, or gouty kidney).

Thrombosis with infarction, or rupture and haemorrhage may occur as sequels. Alternatively death may be due to cardiac failure with all the usual signs.

In cases of malignant hypertension, the renal glomeruli show inflammatory changes resembling those seen in acute nephritis, in addition to the changes already described, the nuclei are increased and leucocytic infiltration is present. Death in these cases is often from uraemia, though it may be due to cardiac failure

SYMPTOMS.—(1) EARLY STAGE. In many cases there may be no symptoms until one or other of the complications appears. This is often the case in placid individuals, in whom the raised blood pressure is frequently found accidentally, for example, on examination for life insurance.

In nervous, highly strung individuals there are often *nervous symptoms* for some time before any of the complications appear. Headaches, dizziness, tinnitus, specks before eyes, fatigue, and palpitation on exertion or on excitement are frequent complaints The symptoms often bear no relation to the height of the blood pressure; this may be higher one week when the symptoms are less severe and vice versa It is questionable whether these symptoms are not due to an independent anxiety state In some cases they only appear *after* the patient has learned that he or she has a high blood pressure and are definitely due to anxiety about blood pressure In some they are associated with the menopause Cases in this group usually have a raised pulse rate, whereas patients with uncomplicated hypertension usually have a relatively slow pulse until cardiac failure or some other complication appears

Occasionally patients take transient attacks of paralysis, known as *angiospastic* or *vasospastic attacks*. There may be a transient hemiplegia or monoplegia, transient ophthalmoplegia with diplopia, or transient blindness. The paralysis

a ring of scar tissue resulting from atheroma; the right kidney was much atrophied while the left was of normal size but showed the changes of malignant hypertension. Though I have witnessed numerous autopsies in cases of primary hypertension, this is the only instance in which any such clue to the aetiology has been found.

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The truth regarding the aetiology of benign and malignant hypertension is probably to be found in the view that multiple factors are capable of causing it. Hypertension should be regarded as a symptom-complex which may arise in various ways but which leads to secondary structural changes.

PATHOLOGY.—The earliest change is a lipoid degeneration in the walls of the arterioles, especially in kidney and retina, but also elsewhere. This is followed by hypertrophy of the arterioles, possibly induced by spasm. As a result of the raised blood pressure, hypertrophy next affects the media of the arteries, especially the aorta, the muscular layers hypertrophy while the elastic lamina splits into several layers. Concentric hypertrophy of the left ventricle appears simultaneously. At a later stage the hypertrophy of the media may be followed by sclerosis and deposition of fibrous tissue (diffuse hyperplastic sclerosis).

ally the haemorrhage is subarachnoid and is not necessarily fatal. Other haemorrhages are less frequent: there may be haematemesis, melaena, haemoptysis, or haematuria. Haemorrhage is especially likely where hypertension is complicated by atheroma or by sclerosis of the media, and the onset may be in relation to some sudden strain or effort which has raised the blood pressure to a still higher level; this is often a matter of medico-legal importance.

Thrombosis. Most often met with are cerebral thrombosis and coronary thrombosis, each of which is frequent. Thromboses elsewhere are much less common, though they occasionally occur in the mesenteric, limb, or pulmonary vessels. Thrombosis is more likely when hypertension is complicated by atheroma, and is apt to occur when the circulation rate is slowest; it frequently develops during sleep. Sometimes it follows strain or severe physical effort, and in these circumstances it is possible that the initial event is an angiospasm which produces sufficient local slowing of the blood stream to precipitate thrombosis. This point may be of importance medico-legally in workmen's compensation cases. In many patients however the precipitating cause of the thrombosis remains obscure, and attacks have been known to develop in almost any circumstances; angiospasm may be responsible.

Cerebral complications include cerebral haemorrhage; cerebral thrombosis (which will be described in Chapter 14); angiospastic attacks; and hypertensive encephalopathy which have been described above.

Hypertensive heart disease sooner or later develops in almost every case, the only exceptions being those who die early in the course of the disease from a cerebral catastrophe and cases of malignant hypertension dying from uraemia. *Angina of effort* is often an early manifestation; it may precede a coronary thrombosis or an acute left ventricular failure by days, by weeks, or by years. *Increasing breathlessness on exertion* sometimes takes the place of angina of effort preceding coronary thrombosis, left ventricular failure or congestive failure. On the other hand, some patients have no warning of approaching cardiac complications until the sudden development of a *coronary thrombosis* or of a *left ventricular failure*. Coronary thrombosis will be described in Chapter 15. Acute left ventricular failure gives rise to an attack of *acute pulmonary oedema*

develops suddenly but clears up rapidly, lasting only a few minutes or a few hours; to begin with, the attacks look like a cerebral thrombosis, but the rapid complete recovery rules out this diagnosis. Occasional patients have an analogous condition affecting the heart in the shape of attacks of *spasmodic angina*. These occur in the absence of effort and at first look like a coronary thrombosis, but the symptoms vanish after anything from 15 minutes up to an hour or two and they leave behind them none of the signs of a myocardial infarct. A third type of attack is described as *hypertensive encephalopathy* (or *hypertensive cerebral attack*), it starts with severe headache, rapidly followed by drowsiness and then coma; there may be muscular twitchings or convulsions while the coma is developing. When comatose, signs of increased intracranial pressure are present—papilloedema, slow pulse, and stertorous respirations, but signs of localised damage to brain in the shape of increased flaccidity in one limb or on one side are absent. The blood urea is normal. These attacks are apt to be confused with uraemia, cerebral haemorrhage, or cerebral thrombosis; the normal blood urea and the absence of localising signs should suggest the diagnosis. The final proof is given by the dramatic recovery in response to lumbar puncture, a less dramatic though still fairly rapid recovery may follow an enema of saturated magnesium sulphate solution, or a hypertonic saline injected intravenously.

Gastro-intestinal symptoms are not infrequent, even in the placid type who show little in the way of nervous symptoms. The most common is a flatulent dyspepsia. In other cases complaint of a feeling of heaviness after food, nausea or vomiting, and anorexia may suggest a chronic gastritis.

(2) LATER STAGE.—The foregoing symptoms or lack of symptoms may be present for months or years before *complications* appear. The complications include *haemorrhage, thrombosis, cerebral vascular accidents, hypertensive heart disease* with its various sequelae, and *renal failure*, one or more of which dominates the picture in the later stages.

Haemorrhage. Epistaxis is the most frequent. Retinal haemorrhages are likewise common, they may or may not cause disturbance of vision. Cerebral haemorrhage occurs in many cases; most often from the lenticulostriate branch of the middle cerebral artery in which case it is rapidly fatal: occasion-

Renal complications. In most cases of hypertension, renal function remains fairly good; although a little albumin may be present in urine with some granular casts and red cells, the blood urea remains normal or only slightly raised and the concentrating power is quite good.

In some cases, however, the picture of the illness is quite different, and is much more like that of a nephritis (*malignant hypertension*) This type is especially apt to occur in young persons—sometimes in the early twenties or even in the late teens, but it may develop in older people; and sometimes [redacted] case starts as a benign hypertension, remains benign and more or less stationary for several years, then suddenly begins to progress rapidly and take on features of a malignant hypertension The earlier symptoms are usually vomiting, dyspepsia, severe headache and malaise with pallor, the blood pressure rises steadily and ultimately reaches very high levels; retinitis and papilloedema are present, albumin casts and red blood cells are *found* in the urine, the blood urea rises progressively and may reach 300-500 milligrammes per 100 c.c. (normal 30-50) [illegible] eks or months, with

[illegible] patient with benign hypertension and kidneys which, though damaged, have been functionally efficient, the fall in blood pressure may determine the onset of renal insufficiency The blood urea rises; headache and vomiting are added to the cardiac symptoms, and convulsions or coma may supervene These cases are sometimes referred to as *cardio-renal failure*.

PHYSICAL SIGNS.—The characteristic physical sign is a *persistently* raised *diastolic* blood pressure, a figure persistently above 90 is suspicious, above 100 is definitely pathological As long as the heart remains efficient, the systolic pressure rises in proportion, usually [redacted] mm. rise in systolic for each 1 mm rise in diastolic Thus starting from 120/80 successive steps are 140/90, 160/100, 180/110, 200/120, 220/130, 240/140, and so on If there is sclerosis of the media, the rise in systolic pressure is proportionately greater, for example from 120/80 to 145/90, 170/100, 200/110, 240/120, etc. If there is cardiac insufficiency, the rise in systolic pressure is proportionately less, examples being 120/80, 135/90, 150/100, 160/110, 170/120, 180/130, with the onset of cardiac failure a blood pressure

the symptoms of which have been described on p 27. This may clear up, or may form the starting-point of *hypertensive heart failure*; in other cases, hypertensive heart failure is ushered in by a period of increasing dyspnoea. The features, which have already been described in greater detail, are pallor, breathlessness, orthopnoea, attacks of cardiac asthma, rapid pulse, soft heart sounds or gallop rhythm, Cheyne-Stokes respiration, signs of congestion of the pulmonary bases, and confusion or delirium which is apt to be worse at night

In some cases there is little drop in blood pressure when left ventricular failure develops; the pulmonary congestion appears to induce a reflex vaso-constriction sufficient to counteract any fall in cardiac output; in some patients there is actually a rise in pressure during an attack of cardiac asthma. Often, however, there is a moderate drop in pressure, usually affecting the systolic to a greater extent than the diastolic; on occasion the pressure falls to such extent that the reading is within normal limits or even below normal. Myocardial pain simulating that of coronary occlusion accompanies the onset of left ventricular failure in some instances; differentiation from coronary occlusion may be possible only when multiple chest lead cardiograms have failed to demonstrate a localised myocardial infarct

If the patient survives the initial acute stages, symptoms of *congestive failure* may be added later—cyanosis and oedema with enlargement of the liver, oliguria, and albuminuria The onset of right ventricular failure is frequently accompanied by considerable relief of the more distressing symptoms; orthopnoea lessens, attacks of cardiac asthma cease and the mental state improves with abatement of restlessness, confusion, and delirium. The relief is no doubt due to lessening of the pulmonary congestion in consequence of failure of the right ventricle, and the symptoms sometimes recur when the right ventricular failure improves. Occasionally the picture is one of congestive failure from the start, with increasing breathlessness followed by oedema. These cases are attributed to massive hypertrophy of the interventricular septum which bulges into the right ventricle, giving rise to mechanical obstruction to the blood flow in the latter ("Bernheim's syndrome"). Most hypertensive patients retain a normal rhythm even when failure is present, but a few develop auricular fibrillation or flutter.

Renal complications In most cases of hypertension, renal function remains fairly good; although a little albumin may be present in urine with some granular casts and red cells, the blood urea remains normal or only slightly raised and the concentrating power is quite good.

In some cases, however, the picture of the illness is quite different, and is much more like that of a nephritis (*malignant hypertension*). This type is especially apt to occur in young persons—sometimes in the early twenties or even in the late teens, but it may develop in older people; and sometimes a case starts as a benign hypertension, remains benign and more or less stationary for several years, then suddenly begins to progress rapidly and take on features of a malignant hypertension. The earlier symptoms are usually vomiting, dyspepsia, severe headache and malaise with pallor; the blood pressure rises steadily and ultimately reaches very high levels; retinitis and papilloedema are present; albumin casts and red blood cells are found in the urine; the blood urea rises progressively and may reach 300-500 milligrammes per 100 c.c. (normal 30-50); uraemia supervenes after a few weeks or months, with convulsions followed by coma, then death.

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have been
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insufficiency The blood urea rises; headache and vomiting are added to the cardiac symptoms, and convulsions or coma may supervene These cases are sometimes referred to as *cardio-renal failure*.

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which has been high may fall so as to appear within normal limits, for example from 180/110 to 135/90 or to 115/85

It must be emphasised that a raised systolic blood pressure without a raised diastolic pressure does not indicate hypertension; such a reading may be due to aortic regurgitation,

FIG 65.—Hypertensive heart disease Female aged 30 Moderately stout (height 5 ft. 5½ in, weight 11 st 2½ lb) History of breathlessness on walking for 8 months and of an attack of "cardiac asthma" shortly after the onset Cardiac apex 4 inches from mid line, sounds of good tone *Blood* pressure 198/104 Electrocardiogram shows left axial deviation, PR = 0 24 second Depressed RT interval in leads 1 and 2, inverted T3, probably a digitalis effect, but the depressed RT1 may be due to left ventricular hypertrophy The X-ray shows a short broad chest with high diaphragm, the aortic shadow is dense, the left ventricular contour prominent, and the lung fields are slightly congested

to thyrotoxicosis, to sclerosis of media, to muscular effort, or to emotion, even the excitement of being examined.

As the diastolic blood pressure rises, the left ventricle hypertrophies. The cardiac impulse becomes forcible and sustained or heaving, and it is usually a little displaced to the left Provided there is no dilatation, considerable hypertrophy may be present with an apex only *4 inches from the mid line, it is*

the character of the impulse rather than its position which provides the clue to concentric hypertrophy. With this, the shape of the heart as seen by X-ray alters, the left ventricular contour becomes more prominent in the antero-posterior and left oblique views (Figs 65 and 66, also Figs. 10 and 12), while the aorta

Fig 66 — Advanced ...
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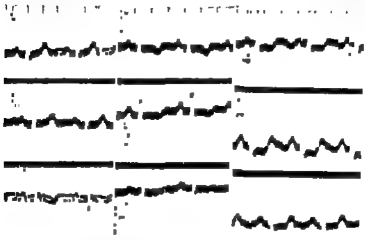
te ... shown in Figs 10 and 12 (early stage with concentric hypertrophy of left ventricle), and Fig 68 (a case of former hypertension in whom sclerosis of the media has developed)

generally becomes wider, often "unfolded", and sometimes tortuous, by "unfolding of the aorta" is meant an increase in the radius of the arc formed by its arch (Fig 64) Left axial deviation usually develops in the cardiogram. At first the heart sounds are of good tone, the second aortic sound being accentuated Later, when cardiac insufficiency sets in, the first sound becomes soft, and is often accompanied by a systolic murmur at the apex The impulse is less forcible and the apex more displaced to the left Radiologically the heart is larger

and congestion is seen in the lung field (Fig. 66, p. 293, and Fig. 10, p. 85). In the cardiogram T1 becomes lower, then inverted (left ventricular hypertrophy, Fig. 67). In the final stages of hypertensive heart failure, gallop rhythm is frequent; it may be presystolic, systolic, or proto-diastolic. Accentuation of the pulmonic second sound due to pulmonary congestion and



A B C

FIG. 67.—CARDIOGRAMS IN HYPERTENSION

A *Hypertension* Left axial deviation T3 inverted Case of old cerebral thrombosis, now becoming stout and breathless

B *Hypertensive heart disease with left ventricular hypertrophy* Left axial deviation. Inversion of T1 and T2 Male, aged 59, angina of effort and attacks of spasmodic angina

C *Hypertensive heart disease with left ventricular hypertrophy* Male, aged 60, with deformed chest, hypertension, and enlarged heart Complaint, angina of effort The axis is normal RT is depressed in leads 1 and 2, indicating left ventricular hypertrophy

pulmonary hypertension sometimes replaces the accentuation of the second aortic sound.

The pulse is usually slow (except in nervous, menopausal, or thyrotoxic cases) until complications supervene; it may remain slow with congestive failure, but is usually rapid with the more common hypertensive failure. The radial arteries may be normal, they may be thickened and wiry (medial hypertrophy), or they may be thick, tortuous, and nodular when medial sclerosis coexists. The same applies to the brachial and other accessible vessels, in some cases the brachial arteries are obviously thickened though the radial arteries are impalpable. Changes are found most constantly in the retinal arterioles. These tend to be narrow in comparison with the veins

and to show irregularity in their calibre; where they cross a vein the latter is nipped and is seen to be distended distal to the crossing. In more advanced stages highly refractile patches appear along the course of the arterioles; these may coalesce so as to give an appearance like silver wire or copper wire. In still more severe cases, especially though not exclusively in malignant hypertension, flame-shaped haemorrhages and glistening white spots ("fish-scale patches") appear in the retina, the condition is then termed *hypertensive retinopathy*. Papilloedema is present in many cases of malignant hypertension; in the latter, and in cases secondary to nephritis there may be in addition woolly white patches of exudate, the term *albuminuric retinitis* being applied

In *benign cases*, the urine may be albumin-free and the renal function normal, there may be an occasional trace of albumin, or a constant trace with some granular casts and a few red blood cells. In malignant cases albuminuria is greater, casts and red cells are more numerous, renal function is poor, and the blood urea is much raised Intermediate types occur. The urinary output is maintained in benign cases and in the earlier stages of malignant cases; it falls with the onset of uraemia or with cardiac failure.

There may be signs of *other complications*, e.g in the central nervous system. Or there may be signs of a *primary disease*

. and slow in progress The complexion is often highly coloured or florid The blood pressure rarely rises very high, the range 180/110 to 220/130 embraces the majority of cases The kidney function remains good Patients frequently carry on for many years before developing complications. Death ultimately results from a cardiac or cerebral complication

Malignant hypertension is more rapid in onset and progress. The complexion tends to be pale rather than florid. The blood pressure rises steadily and often reaches very high levels, for example from 260/140 to 300/180 Albuminuria is constant and renal function impaired with rising blood urea. Retinitis is usual and papilloedema frequent, some physicians regard retinitis, others papilloedema, as the criterion which distinguishes malignant from benign hypertension, but many refuse to accept such a narrow distinction as absolute. Death

in most cases results from uraemia, though sometimes from cerebral or cardiac complications. In young patients the illness resembles a nephritis more or less closely. The duration varies from a few weeks to a few months or occasionally a year. In older patients this is still the case in the majority, but cerebral and cardiac complications are by no means infrequent. Cerebral vascular lesions are often multiple. The complications develop earlier than in benign hypertension.

The cardiogram in malignant hypertension occasionally fails to show left axial deviation despite a very high blood-pressure reading and an enlarged heart. The QRS complexes are sometimes abnormal.

Some cases of hypertension intermediate between the benign and malignant types show alternating active and quiescent phases. During the active phases the patient is pale, the pulse rate elevated, the disease appears progressive and complications are frequent During quiescent phases the patient is of normal colour or florid, the pulse rate is normal, the disease appears stationary, and complications are infrequent

TREATMENT.—It is an invariable rule that the treatment should be based on a consideration of *the patient and his general condition, not on a sphygmomanometer reading.* In certain suitably selected cases surgical procedures hold out hope of cure (p. 300); but many cases are unsuitable for surgical treatment. The general principles of medical treatment will be described first.

Benign hypertension When raised blood pressure is found accidentally, the first question is whether the patient should be told. *Often more harm than good is done by telling him*, especially in highly strung or nervous patients; if the patient's habits and mode of life are sound and if his case does not fall into one of the categories suitable for surgical procedures there is no curative treatment for the hypertension, and there is nothing to be gained by alarming him. A highly strung patient should not be informed of accidentally found hypertension unless it is necessary in order to induce him to modify his habits, or to undergo curative treatment. It may be wise in some cases to tell the patient's husband or wife, if the findings suggest that complications are likely in the near future; in deciding whether to adopt this safeguard, the temperament of the person who is to be told must be taken into account

Enquiry should be made into the patient's mode of life and habits. Any excesses in the matter of food, drink, physical strain, or mental stress must be corrected. It is important to remember that patients require a certain amount of exercise to keep them fit; insufficient exercise leads to a sluggish circulation and increases the risk of thrombosis—it can be nearly as harmful as excessive exercise. The height of the blood pressure is not the indication for restricting exercise (unless it is very high in a patient with brittle arteries); it is the capacity of the heart which determines any restriction necessary, and the restriction should be the minimum which is required. Occupation is essential to prevent patients from becoming morbidly introspective—a nuisance to themselves, their friends, and their doctor; as long as the patient is able for his own work he should be encouraged to carry on; if his usual occupation is unsuitable he should be persuaded to take up some other work which is within his capacity. Similar considerations apply to recreations and games. Finally, sleep is essential to health; adequate sleep must be ensured; if necessary sedatives such as phenobarbitone should be used.

Any general condition which requires treatment should be dealt with. In cases with *nervous symptoms*, these are best controlled by sedatives, such as ½ grain of phenobarbitone or 15 grains of ammonium bromide thrice daily. "Theominal", a combination of ½ grain of phenobarbitone with 5 grains of theobromine is popular and useful; its chief benefit is probably attributable to its phenobarbitone content, though the diuretic effect of the theobromine is no doubt of some value. None of these drugs will affect the patient's basic blood pressure level; what they will do is to diminish or abolish nervous tension, and they will reduce the blood-pressure reading only in so far as it has been raised above its basic level by nervous stress. Reassurance and a good prognosis (when justified) will often produce a similar result with symptomatic improvement.

Obesity is common in hypertensive patients, and they usually feel better if their weight is reduced. The total calories in the diet, and especially calories derived from carbohydrates and fats are restricted. A reduction in weight is sometimes followed by a slight lowering of the blood pressure. A low sodium diet (p. 450) will reduce the blood pressure in some

patients irrespective of the presence or absence of obesity; the effect is maintained only so long as the diet is continued.

Menopausal symptoms should be treated by oestradiol (stilboestrol) and sedatives.

Myxoedema. Hypertension does not contra-indicate the use of thyroid, nor do cardiac symptoms contra-indicate thyroid unless trial shows that they are aggravated by it, while this sometimes happens, quite often the cardiac symptoms are improved. Therefore the effect of thyroid should be tried beginning with a small dose (½ grain daily), gradually increasing it until the myxoedema is controlled or until the dose is found beyond which cardiovascular symptoms become aggravated.

Diabetes. Should be treated as in a non-hypertensive case, with this difference. Ketosis must be avoided, but hypoglycaemia should not be risked. *It is preferable to keep the patient with a trace of sugar in the urine than to make him completely sugar-free. Many elderly hypertensive diabetics do best on* dietetic treatment alone, *insulin should not be used in such* cases except when ketosis cannot be prevented without it.

Nephritis. When hypertension is secondary to nephritis, and if renal function is impaired, protein should be restricted in the diet to the minimum amount required to replace daily wear and tear—i e about 60-70 grams daily. It is immaterial whether the protein is given as red or white meat; the ban on red meat which is so popular is merely a fetish. Nephritic patients should be advised to drink ample water—a glass night and morning in addition to what they normally take with their meals; *this aids the renal compensating mechanism. Constipation* should be avoided, but patients should not be purged. Restriction of salt in the diet is often helpful.

Gout demands restriction of purin-containing foods.

Treatment of complications. Cardiac complications. Angina of effort or dyspnoea on exertion is treated by suitable restriction of activity; coronary thrombosis, cardiac asthma, or acute pulmonary oedema by rest with sufficient morphia to relieve distress; hypertensive heart failure by rest, sedatives, theophylline-ethylene-diamine ("Cardophylin"), digitalis, and mercurial diuretics, congestive failure by rest, mercurial diuretics and digitalis. Details are given in Chapters 15 and 22.

Cerebral complications. Angiospastic attacks are transient and pass off without treatment, sedatives will often prevent

their occurrence or reduce their frequency. Hypertensive encephalopathy responds rapidly to lumbar puncture, which is the treatment of choice; less certain and less rapid is a saturated magnesium sulphate enema or an intravenous hypertonic saline Cerebral thrombosis and cerebral haemorrhage demand rest and nursing along the usual lines, and if the patient survives, treatment of the resulting hemiplegia, or other disability.

Haemorrhage Epistaxis is usually beneficial up to a point—it may well prevent a more serious haemorrhage elsewhere; as a general rule it may be allowed to continue for a time before the nose is plugged. Other haemorrhages are treated on general lines, for example rest and morphine for haematemesis or haemoptysis

Thrombosis The treatment of superficial thrombosis has been described on pp. 38 and 40, while that of cerebral or coronary thrombosis is discussed in Chapters 14 and 15. Thrombosis in inaccessible vessels elsewhere is treated on the same lines as emboli in inaccessible vessels (p 38).

Lowering blood pressure A fall in blood pressure may precipitate thrombosis in atheromatous patients, or renal insufficiency in those with damaged kidneys. Except in emergency, blood pressure should not be lowered without first excluding gross atheroma and seriously impaired renal function. Even then it should not be lowered merely *for the sake of* doing so, but only for definite indications, which include .

(a) Recurrent or persistent headache , severe exacerbation of headache associated with a temporary rise in pressure above its usual level

(b) Hypertensive cerebral attacks , cerebral angiospastic attacks associated with a temporary rise of pressure; retinopathy

(c) Impending heart failure , effort angina; spasmodic angina associated with a temporary rise of pressure.

The *nitrites* will produce a fall of pressure which develops rapidly, but is very transient, passing off within a few minutes They may be given in the form of amyl nitrite (3 minims in capsule, the capsule to be broken and the vapour inhaled) , this is the most rapidly acting form , alternatively a tablet of trinitrine grain $\frac{1}{100}$ should be chewed and swallowed The nitrites are suitable for spasmodic angina.

The best means of producing a more lasting fall in pressure is to put the patient completely at rest in bed and give sedatives. Any fall so produced is safe but slight. A further fall in pressure may be achieved by means of venesection. A saturated magnesium sulphate enema will also produce a little lowering of pressure, as will a purge.

Sodium sulphocyanate and *potassium thiocyanate* have been recommended. Dosage is controlled by estimation of the serum thiocyanate, at first weekly, later fortnightly or monthly. An initial dose of 0 1 gm is given thrice daily till a level of 5 to 8 mg. per 100 ml. in the serum is attained; if there is no improvement, dosage may be pushed till the serum level is 8 to 12 mg per 100 ml. Opinions differ as to its efficacy. Advocates claim alleviation of headache and dizziness with a fall in blood pressure Toxic effects include rash, malaise, asthenia, nausea, indigestion, pains in the limbs, and impotence. Severe renal damage is generally held to contra-indicate thiocyanates, but Watkinson and Evans claim occasional benefit even in uraemic cases. Patients with myocardial disease derive less benefit Some patients respond well to thiocyanate after an unsuccessful lumbo-dorsal sympathectomy.

Tetra-ethyl-ammonium bromide (T E A B), *pentamethonium bromide*, and *hexamethonium bromide* act by blocking impulse transmission in the sympathetic ganglia. They are used to determine the stability or lability of a raised blood pressure and as a guide to the probable effect of sympathectomy, in addition to or in place of the sodium amytal test (p. 302). T E A.B has unpleasant side effects and is unsuitable for treatment, penta- and hexamethonium bromide are on trial

Surgical treatment of hypertension Cure of hypertension can be achieved in certain groups of cases by suitable surgical procedures.

Nephrectomy. Hypertension secondary to unilateral kidney disease is sometimes curable by removal of the affected kidney Although these cases are rare, the possibility should be considered both in benign and malignant hypertension if there is a history of cystitis or pyelitis, if there is no hypertensive family history, or if the patient is young. Microscopic examination of the urine with intravenous or retrograde pyelography will usually settle the diagnosis. The aim should be to operate before irreversible damage has occurred in the heart, arteries,

brain, or remaining kidney; the function of the latter should be assessed separately by ureteric catheterisation coupled with urea clearance or concentration test. Where a family history of hypertension is obtained, a successful result is less certain; the presence of unilateral kidney disease does not preclude development of an independent hereditary essential hypertension.

Adrenalectomy Hypertension secondary to phaeochromocytoma is curable by removal of the tumour. Though usually paroxysmal at first, the hypertension is sometimes persistent from the start; cold, pale extremities are suggestive of hyperadrenalism Roth and Kvale's histamine test is valuable in paroxysmal cases, while Goldenberg's benzo-dioxane test has diagnostic value with persistent hypertension (see Chapter 17, p. 370) Adrenalectomy has been tried on occasion in cases without a definite tumour, and some good results have been obtained, but the mortality is high.

Lumbo-dorsal sympathectomy. The Smithwick operation involves removal of the sympathetic chain from the 9th dorsal to the 2nd lumbar ganglion on each side with the greater and lesser splanchnic nerves More extensive sympathectomies extend as high as the 4th dorsal ganglion and as low as the 3rd lumbar The operation is followed by symptomatic relief in about 50 per cent of cases, and by a fall in blood pressure to normal levels in a slightly smaller percentage, retinopathy improves and cardiac hypertrophy may regress The fall in pressure is rarely permanent, within 3-5 years a number of patients have once more become hypertensive, and few retain a normal pressure in 5-10 years The immediate mortality of the operation varies from 0 to about [illegible]5 per cent in selected cases of benign hypertension, in malignant hypertension it approaches 20 per cent The results in malignant hypertension are less satisfactory. the blood pressure may drop slightly or remain unaffected. there is often symptomatic relief for a time and prolongation of life for a year or two Sympathectomy is sometimes followed by severe neuralgia, in other cases it gives rise to postural hypotension, both these sequelae tend to subside gradually in from 3 to 6 months.

Sympathectomy is not a *permanent* cure, and is not justified in the early stages of a mild benign hypertension. The *ideal* stage is when blood pressure remains at levels above 200/110 despite medical treatment while changes in the retinal arterioles

The best means of producing a more lasting fall in pressure is to put the patient completely at rest in bed and give sedatives. Any fall so produced is safe but slight. A further fall in pressure may be achieved by means of *venesection*. A saturated magnesium sulphate enema will also produce a little lowering of pressure, as will a purge.

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and cardiac hypertrophy are still slight; it should be advised before advanced changes develop. Best results are obtained in patients under 50. Retinopathy or symptoms of early cardiac insufficiency provide an urgent indication for operation. Arteriosclerosis, arterial calcification, cardiographic evidence of coincident coronary disease, or severe impairment of renal function are contra-indications. The mortality rises with increasing impairment of renal function or with increasing severity of cardiac failure; but some good results have been claimed even in cases with intractable heart failure.

The probable effect of operation can sometimes be gauged by a sedation test · 3 grains of sodium amytal are given hourly for three doses; blood pressure is estimated hourly from the first dose for 12 hours. The lowest readings are obtained during sleep, from 5 to 9 hours after the initial dose. If the pressure falls to normal levels with this test the chances of a satisfactory response to operation are good; if little or no fall in pressure occurs with the test the prospects with operation are less favourable though not necessarily absent.

Malignant hypertension. This is a much more serious disease, and unfortunately the prospects of curative treatment are remote; even the chances of delaying the course of the illness are poor. The general lines of diet are those suitable for hypertension secondary to nephritis, namely restriction of protein and salt, and additional water to drink, constipation should be avoided by gentle laxatives without purging. At a relatively early stage the patient is confined to bed The possibility that the hypertension is the result of unilateral renal disease should be considered, though it is very rare, and if proved, the affected kidney should be removed. In other cases, lumbo-dorsal sympathectomy is probably worth a trial in view of the fact that no other form of treatment offers any prospect of a cure, but the operative mortality is high Apart from the foregoing points, treatment is mainly a matter of nursing and dealing with complications as they arise

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CHAPTER 14

ARTERIAL DISEASE (*contd.*): CEREBRAL VASCULAR LESIONS

CEREBRAL vascular lesions are sufficiently frequent as complications of cardiovascular disease, and especially of chronic arterial degeneration, to warrant a description of their symptomatology and differential diagnosis in a book dealing with disease of the circulatory system.

AETIOLOGY

Three varieties of cerebral vascular lesion occur in cardiovascular disease, viz. embolism, thrombosis, and haemorrhage. Of these, thrombosis is the most frequent. The three occur in different circumstances

Embolism.—Embolism is secondary to one or other of the following acute ulcerative or subacute bacterial endocarditis, from vegetations on the aortic or mitral valve cusps; auricular fibrillation, from intra-auricular thrombosis; coronary thrombosis, from intraventricular thrombosis; aneurysm of the ascending aorta from thrombosis in the sac ; or to venous thrombophlebitis in a patient with atrial or ventricular septal defect (paradoxical embolus) Embolism is rare in acute simple endocarditis Emboli may occur at any age and in either sex, but since endocarditis is the most common cause, the majority affect persons in early adult life or middle age. In some cases the occurrence of an embolism is the first evidence of bacterial endocarditis.

An embolus which lodges at the bifurcation of the internal carotid artery is usually rapidly fatal. Smaller emboli [illegible]

[illegible] to an area of softening; the symptoms are those of sudden loss of function of the affected part of the brain Occasionally an embolus lodges in the central artery of the retina, and it produces sudden loss of vision of one eye with a characteristic appearance on ophthalmoscopic examination

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the ..., lodging in a small branch of one of the cerebral vessels In this case infarction of the corresponding area of the brain occurs, leading to an area of softening, the symptoms are those of sudden loss of function of the affected part of the brain Occasionally an embolus lodges in the central artery of the retina, and it produces sudden loss of vision of one eye with a characteristic appearance on ophthalmoscopic examination.

Thrombosis is most likely to develop in vessels which are the seat of atheroma. Hence it is more frequent in patients beyond middle age who have evidence of atheroma elsewhere, often in the coronary vessels or aorta. Thrombosis is not necessarily associated with hypertension, and in many cases the blood pressure is normal; but atheroma, sclerosis of the media, and hypertension often coexist. Thrombosis is most likely to develop when a fall in blood pressure takes place; many attacks start during sleep; in others vascular spasm is probably the precipitating factor. Thrombosis affecting a large vessel such as the internal carotid causes sudden death. In the case of a smaller vessel infarction and softening of the brain result with symptoms of sudden or gradual loss of function. The central artery of the retina is a seat of thrombosis in occasional cases. Thrombosis sometimes affects a small portion or branch of a vessel in the first instance, extending into collateral branches later; in such a case the onset of symptoms is very gradual, extending over several days, or the progress of the condition may be marked by recurring episodes, each representing an extension of the process. Multiple thrombotic lesions are not uncommon and give rise to the picture of "cerebral softening" or "cerebral arteriosclerosis".

Haemorrhage.—The most important cause of haemorrhage is sclerosis of the media, particularly if associated with atheromatous ulceration and hypertension. Haemorrhage is therefore most common in late adult life (age 50 onwards), and in patients with hypertension and hardened arteries. The lenticulostriate branches of the middle cerebral artery are the most frequent to rupture; the effused blood ploughs up the surrounding brain tissue, destroys the motor tract in the internal capsule, and in many cases escapes into the third ventricle, staining the cerebrospinal fluid. In these cases the haemorrhage is generally fatal. Large haemorrhages into the cerebellum are less frequent; the effused blood may escape into the fourth ventricle, here also a fatal result is usual. At times the rupture affects one or other of the vessels at the base of the brain, blood escaping into the subarachnoid space; the clinical picture is that of subarachnoid haemorrhage, and the result is not necessarily fatal. Smaller vessels sometimes rupture elsewhere, giving rise to localised lesions; rupture of

a pontine artery is commonly fatal, but recovery may follow small cortical lesions.

Intracerebral haemorrhage is a rare result of syphilitic arterial disease. In younger individuals, intracerebral haemorrhage is occasionally met with as a manifestation of one of the haemorrhagic diseases such as scurvy, aplastic anaemia, leukaemia, malignant thrombocytopenia, or haemophilia. It has been described from venous congestion during a paroxysm in whooping-cough. Erosion of a blood vessel by a tumour is another cause of haemorrhage, and in some cases the haemorrhage is the first evidence of the presence of a tumour.

Congenital defects of the muscle coat of the vessels occur in some individuals, the weak part of the vessel expands into a small aneurysm (Congenital or "Berry" aneurysm). They occur especially in the vessels forming the Circle of Willis, but are also found occasionally in the intracerebral vessels. Rupture of such an aneurysm is an occasional cause of an apparently primary cerebral haemorrhage in a young person

Subarachnoid haemorrhage.—In patients beyond middle age, subarachnoid haemorrhage is usually secondary to atheroma or to hypertension In younger patients, rupture of a congenital aneurysm is the usual cause. Other causes include the haemorrhagic diseases, and trauma

In cases of congenital aneurysm, in place of sudden rupture with diffuse haemorrhage a gradual leakage of blood sometimes takes place, this gives rise to a characteristic symptom-complex due to pressure from the resulting haematoma.

Extradural haemorrhage is the result of bleeding from the meningeal vessels, and is usually the result of trauma Fracture of the skull is not a necessary preliminary, haemorrhage can occur while the skull remains intact The resulting symptom-complex is characteristic ("extradural" or "subdural haematoma").

Localised capillary haemorrhages arise from "contre-coup" in head injuries and are probably responsible for the symptom-complex of *cerebral contusion*

Pathology and Pathological Physiology

A vascular lesion, unless rapidly compensated by development of a collateral circulation, results in destruction of a

certain area of brain tissue ; the affected area is either infarcted, or torn up by effused blood. An area so destroyed is permanently damaged ; no regeneration of nerve cells occurs within the central nervous system. If the original lesion is not fatal, the affected area undergoes softening, and ultimately *forms a cyst containing pale yellowish fluid.*

Surrounding the area which is actually destroyed, a further zone of brain tissue is affected, by partial anoxaemia and later by inflammatory reaction in the case of embolism or thrombosis, or by pressure in the case of haemorrhage. This area exhibits temporary loss or diminution of function , the nerve cells and fibres are not destroyed, and they can recover their function if the patient survives. To this extent recovery of function is possible after a vascular accident Thus if the zone of destruction includes a portion of the motor tract, the resulting paralysis is permanent ; but if the motor tract is merely included in the surrounding zone of partial anoxaemia or pressure the paralysis is temporary, and complete recovery from it is possible

Symptoms

The symptoms depend on the situation, the size, and to a less extent on the nature of the lesion. In embolism, the onset is instantaneous , but this does not preclude the possibility of subsequent thrombosis of the blocked vessel with extension into a proximal collateral branch and development of fresh symptoms at some later period With haemorrhage the onset is usually rapid, albeit not quite so abrupt as with embolism , the symptoms quickly become more severe and more widespread. In the case of thrombosis the onset is much more variable, sometimes rapid, sometimes very gradual, and sometimes in stages by jumps In any case the situation and size of the lesion determine the clinical syndrome

(1) *Sudden death* may occur with lesions affecting a large vessel such as the internal carotid.

(2) *Apoplexy or stroke.* In many cases the attack is preceded by symptoms due to sclerosis of the media or to hypertension. The cerebral symptoms tend to be more severe for a time before the actual onset , headache, tinnitus, or vertigo are more intense. In hypertensive cases a rise of pressure may determine the actual rupture, the attack being brought on by

exertion, by a fit of coughing, or by emotion; but it may occur suddenly, without warning and without any demonstrable exciting cause. In atherosclerotic cases a fall of pressure is liable to precipitate the attack, which often starts during sleep.

The onset of the "stroke" is associated with sudden or relatively sudden loss of consciousness. In some cases the victim is suddenly struck unconscious without warning. In others there is intense headache, faintness, dizziness, nausea, vomiting, or an epileptiform convulsion; unconsciousness follows often in a matter of minutes but sometimes not for an hour or longer. When the coma is relatively slow in developing, the patient may complain of localising symptoms such as slurring of speech, greater weakness in one arm, one leg, or one side of the face, paraesthesiae, or a visual defect.

Immediately after the onset, the patient is collapsed and shocked, with a rapid feeble pulse, often barely perceptible. Some die within a few minutes of the onset, but if not, the pulse soon becomes full and bounding.

At this stage the patient is completely unconscious, and he cannot be roused. The face is flushed. The respirations are stertorous or snoring, the cheeks being frequently puffed out with each expiration and drawn in on inspiration. If the facial portion of the motor area is involved, one side of the face will be more flaccid and the angle of the mouth will droop on that side. In some cases there is conjugate deviation of the head and eyes; the muscles moving the head *and eyes to the side* opposite the lesion are paralysed and the head and eyes consequently deviate towards the side of the lesion. The pupils vary; they are often dilated in cerebral haemorrhage, and they may be unequal; in pontine haemorrhage they are small ("pin-point pupils"). The limbs are flaccid at first and the reflexes are absent; *greater flaccidity* on one side points to a lesion of the opposite side of the brain involving the motor cortex or motor tract.

Once the initial collapse and shock have passed off, or in some cases right from the onset, the limbs cease to be flaccid, becoming spastic instead. The tendon jerks return and are spastic in type; the plantar responses return and are extensor. As recovery occurs, spasticity disappears and the plantar responses become flexor, but often one limb or one side remains spastic with extensor plantar response and clonus,

certain area of brain tissue; the affected area is either infarcted, or torn up by effused blood. An area so destroyed is permanently damaged; no regeneration of nerve cells occurs within the central nervous system. If the original lesion is not fatal, the affected area undergoes softening, and ultimately forms a cyst containing pale yellowish fluid.

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aphasia, hemianopia, cerebellar vertigo, dysarthria or dysphagia, or in some cases mental symptoms such as confusion or disorientation. Symptoms which have arisen in this way may be permanent or transient, depending on whether the area of brain responsible for them is actually destroyed or merely affected by pressure or anoxaemia from a neighbouring lesion. In the case of transient symptoms the duration varies from a few hours up to a few days. Symptoms which merely last a matter of minutes are more likely to be due to angio-spasm than to an organic vascular lesion, though the differentiation of these conditions is sometimes impossible.

(4) *Multiple vascular lesions.* Multiple thrombotic episodes are not uncommon in patients with atherosclerosis and hypertension The picture may be one of mental deterioration ("organic reaction type") with impairment of memory, intellect, and emotional control, resulting in childish behaviour; with this there are sometimes localising symptoms. The occurrence of sub-thalamic lesions leads to a picture resembling the Parkinson syndrome in some cases. Bulbar lesions of vascular origin simulate progressive bulbar paralysis ("pseudo-bulbar paralysis") Occasionally the clinical picture suggests uraemia ("pseudo-uraemia") The symptoms often develop gradually and progress slowly, or one group of symptoms may appear suddenly, to be followed after an interval by another group

(5) *The posterior inferior cerebellar syndrome.* Lesions of these vessels are less infrequent than those of the superior cerebellar arteries, and they produce a characteristic symptom-complex due to involvement of the cerebellar tracts, the long sensory tracts, and the upper medullary nuclei (facial, vestibular glosso-pharyngeal, and often vagus); the long motor tracts lying anteriorly are rarely involved The ...

... and loss of muscle tone affect the arm and leg on the side of the lesion Nystagmus is seen ... slow movement ...

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indicating an upper motor neuron lesion; and one side of the face may remain paralysed. During the period of coma, control of the bladder is lost; distension is followed by "retention-overflow", that is to say the bladder fills up to a point, then partially empties. The bowels may remain constipated, or there may be incontinence of faeces.

If the motor tracts are not destroyed, but instead are irritated by a neighbouring lesion, muscular twitchings of convulsive type take the place of the upper motor neuron paralysis; these may affect the face, head, arms, or legs, or may be generalised.

A sudden fall of temperature suggests rupture of a haemorrhage into the lateral ventricle, and is an ominous feature. A rapidly rising temperature is usually an indication of pontine haemorrhage and is commonly followed by death within a few hours. *Haemorrhage near the medulla or into the fourth* ventricle may cause glycosuria and simulate diabetic coma. In severe cases the pulse and respiration rates are rapid; flushing is intense and sweating sometimes profuse, lividity appears, accompanied by tracheal rale; the pulse becomes weaker, and death follows. Alternatively the patient develops a secondary bronchopneumonia and dies after a few days.

In favourable cases coma persists for a variable period, from a few hours up to a few days. Consciousness gradually returns, and, once it has been regained, symptoms of the local lesion remain. These depend on the site of the lesion, there is frequently a hemiplegia, but sometimes a monoplegia, a facial paralysis, a hemianopia, a defect of speech, or a cerebellar syndrome; *in fact any localisation is possible.* The residual symptoms may be permanent, or they may gradually improve, sometimes they disappear entirely. Slow improvement continues over a period of several weeks, and sometimes for two or three months, any symptoms which persist at the end of six months are almost certainly permanent.

(3) *Lesions of smaller vessels, insufficient to cause coma.* These are more often due to embolism than to thrombosis or haemorrhage, though they can occur in any of the three conditions. The symptoms come on either instantaneously (embolism) or comparatively rapidly (thrombosis); in the latter case their development may be a matter of a few minutes or a few hours. The actual symptoms depend entirely on the situation of the lesion; they include a hemiplegia or monoplegia,

oedema; in some cases haemorrhages are seen in the retina; they are larger than the flame-shaped haemorrhages of a hypertensive retinitis and are apt to be more irregular in shape. On lumbar puncture, the cerebrospinal fluid is found to be under increased pressure, and it is intimately mixed with blood if performed early, later it is yellowish in colour owing to the presence of altered blood pigment Apart from blood cells there is no increase in cell content, such polymorphonuclears as are present being proportional to the amount of blood. Culture reveals a sterile fluid Transient albuminuria is usual, irrespective of the cause of the haemorrhage.

A large subarachnoid haemorrhage may prove fatal. Otherwise improvement commences in a few days and recovery is usually complete within two to three weeks. In cases due to hypertension, the latter is usually of the malignant type, and the prognosis is unfavourable, even though recovery from the haemorrhage occurs In cases due to rupture of a congenital aneurysm, the prognosis is more favourable; future recurrences are likely and while one of them may prove fatal, there is often a prolonged interval of good health between them.

The differential diagnosis in this type of case is from meningitis, in which the onset is less sudden and is often preceded by fever and symptoms of a general infection, the cerebrospinal fluid is not intimately mixed with blood, but contains excess of leucocytes and yields the causative organism on culture or with appropriate staining methods

(B) *Gradual leakage from a congenital aneurysm, subarachnoid haematoma* This is an alternative clinical syndrome in cases of bleeding from a congenital aneurysm Once again the patient is usually a young adult, and free from other signs of cardiovascular disease. The symptoms consist of intense headache followed by signs of cranial nerve palsy In most cases the oculo-motor nerves are chiefly affected, leading to a complete ophthalmoplegia with ptosis and absence of light reflex, this may be unilateral or bilateral

reflex

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...ing's sign The intensity of the headache is apt to vary from time to time, and during

side as the spino-thalamic fibres from the arm and leg have already crossed in the cord. Dysphagia and dysarthria result from involvement of the motor nuclei; and if the lesion extends sufficiently high, there may be an infranuclear facial paralysis. Death may occur from extension to the area of the respiratory centre, or from a secondary pneumonia; but in the absence of these complications complete recovery may occur.

(6) *Pontine haemorrhage.* The attack begins like an apoplexy with sudden loss of consciousness and paralysis of both arms and both legs; the plantar responses are absent or bilaterally extensor. The pupils are very small ("pin-point pupils"). The temperature rises rapidly and hyperpyrexia occurs before death, which usually takes place within 12 to 24 hours.

(7) *Mid-brain haemorrhage or thrombosis* (posterior cerebral artery, superior cerebellar arteries). The chief localising symptoms result from involvement of the oculo-motor nuclei, which leads to ptosis, loss of accommodation and light reflexes, and squint or diplopia. If the connections between the two sides are affected, the eye symptoms are bilateral, and they resemble those produced by a pineal tumour. With forward extension of the lesion, the motor tracts may be affected. The diagnosis from pineal tumour can be made on the ground of sudden onset in a person with cardiovascular disease of a type which predisposes to vascular accidents.

(8) *Subarachnoid haemorrhage.* In many cases the patient is a young adult and the haemorrhage is attributable to rupture of a congenital aneurysm. In older patients subarachnoid haemorrhage may be a complication of hypertension or atherosclerosis, though this is a less common situation for the rupture than within the brain tissue. The onset is sudden, with headache, followed by signs of meningeal irritation and of increased intracranial pressure. The headache is often occipital and accompanied by pain in the back of the neck, but it may be frontal or generalised. There is usually rigidity of the neck with a positive Kernig's sign, and sometimes head retraction. Evidence of cranial nerve paralysis occurs in some cases. The headache is severe and persistent. Soon it is followed by drowsiness or unconsciousness, and there may be vomiting as the coma is developing. The pulse is normal in rate or slow. Ophthalmoscopic examination shows the presence of papill-

oedema; in some cases haemorrhages are seen in the retina; they are larger than the flame-shaped haemorrhages of a hypertensive retinitis and are apt to be more irregular in shape. On lumbar puncture, the cerebro-spinal fluid is found to be under increased pressure, and it is intimately mixed with blood if performed early; later it is yellowish in colour owing to the presence of altered blood pigment. Apart from blood cells there is no increase in cell content, such polymorphonuclears as are present being proportional to the amount of blood. Culture reveals a sterile fluid. Transient albuminuria is usual, irrespective of the cause of the haemorrhage

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the periods when it is more intense there may be drowsiness with other manifestations of increased intracranial pressure. The cerebrospinal fluid varies; at times it may be blood-stained, at others yellow; if the effused blood is shut off from the general subarachnoid space by adhesions, the fluid may appear normal.

(10) *Extradural haemorrhage.* The haemorrhage occurs from the middle meningeal artery or one of its branches, and is nearly always traumatic in origin; vascular sclerosis increases the probability of rupture in consequence of an injury which might otherwise fail to cause haemorrhage. The effused blood accumulates between the skull and the dura mater, giving rise to symptoms simulating those of a cerebral tumour or cerebral abscess. The usual history is that the patient has had a head injury, generally associated with concussion though not necessarily with fracture of the skull; in some cases he is merely momentarily dazed but not unconscious. As the immediate concussion passes off, consciousness returns and the patient may appear normal for a day or two, or he may complain of headache at the site of the injury. In the course of three or four days the headache becomes more severe and the patient begins to be apathetic and drowsy, gradually he becomes semi-comatose or comatose. There is often vomiting. The pulse is slow and there may be papilloedema. In some cases there are signs of localised pressure on part of the cortex corresponding to the site of the haematoma; often these take the form of a hemiplegia or a monoplegia in view of the usual site of the bleeding. In cases where the bleeding is more profuse, the period of apparent recovery from the initial concussion is lacking; the symptoms of compression follow immediately on the injury. The cerebrospinal fluid is normal in cases where the haemorrhage remains outside the dura, if there is a tear in the dura, the fluid contains blood. The diagnosis is usually clear from the history; in the absence of a history, cerebral tumour, cerebral abscess, subarachnoid haemorrhage and intracerebral haemorrhage must be excluded. The treatment is surgical.

Differential Diagnosis of Cerebral Vascular Lesions

There are three steps in the diagnosis of vascular lesions; first, recognition that the lesion is vascular as opposed to

inflammatory or neoplastic; second, recognition of its site; and third, recognition of its nature (i.e. embolic, thrombotic, or haemorrhagic).

The distinction between a vascular lesion and inflammatory or neoplastic lesions is aided by a history of the onset. Vascular lesions are usually more sudden in onset; but thrombosis sometimes develops gradually over a period of a few days, and on the other hand a tumour hitherto silent sometimes erodes a vessel, causing haemorrhage as the first evidence of its existence. Examination of the cardiovascular system will reveal evidence of pre-existing cardiovascular disease of one of the types mentioned in discussing the aetiology; in the case of congenital aneurysms, however, the remainder of the cardiovascular system is commonly normal. The cerebrospinal fluid is often helpful. uniform admixture with blood points to a haemorrhage. It must be noted, however, that blood-staining of the fluid may occur accidentally from puncture of a vein during the process of lumbar puncture, in this case the admixture with blood is not uniform; the first few drops may be deeply blood-stained, becoming less so as the fluid continues to flow, or the first few drops may be clear, followed by the appearance of blood. Again, blood is not always present in the cerebrospinal fluid in cases of haemorrhage, haemorrhage into the brain substance must rupture into the third or fourth ventricle before blood will appear in the fluid. Absence of blood with an increased cell content points to an inflammatory lesion, yet some cases of thrombosis lead to an increased cell content if the area of softening involves the surface of the brain. An increased protein content may be found with vascular, inflammatory, or neoplastic lesions.

... careful consideration of history, clinical features and cerebrospinal fluid findings is required.

The question of localisation of vascular lesions differs in no way from the localisation of other cerebral lesions. The symptoms and signs associated with the various vascular syndromes have been described.

The differentiation between thrombosis, embolism, and haemorrhage depends on a consideration of the onset and of the

type of cardiovascular disease from which the patient is suffering. Examination of the cerebrospinal fluid is of assistance in differentiating haemorrhage from the others. In cases of congenital aneurysm, the condition can sometimes be demonstrated by arteriography, that is, by X-ray after the injection of a radio-opaque dye into the internal carotid artery, the procedure is not entirely without danger and should be reserved for cases in which it is essential. A diagnosis of embolism requires demonstration of a possible source. Though recovery from a small haemorrhage is possible, a massive haemorrhage is invariably fatal; cases which recover are more likely to be due to thrombosis or embolism than to haemorrhage. Other considerations which aid the differentiation have been discussed under the headings of "aetiology" and "symptoms".

Treatment of Cerebral Vascular Lesions

During the acute stage, treatment is mainly a matter of nursing. The unconscious patient must be kept quiet, if noisy, or if taking convulsions, sedatives will be required. Care must be taken to avoid burns from hot-water bottles. Pressure points should be treated with spirit, and the patient's position should be changed from time to time to prevent the development of bed sores. The bladder should be examined at regular intervals, and should be catheterised whenever distended, preferably before spontaneous emptying occurs; when incontinence takes place without much distension, a catheter must be tied in. If the period of coma is prolonged, tube feeding or rectal feeding is necessary.

Lumbar puncture is mainly of diagnostic value, but in cases of subarachnoid haemorrhage, and especially when there are signs of increased intracranial pressure, lumbar puncture has considerable therapeutic value. In subarachnoid haemorrhage it should be repeated every other day or every day, according to the severity of the symptoms. With the other vascular lesions, lumbar puncture has no therapeutic value. Formerly venesection was much practised in the belief that by lowering the blood pressure it might help to arrest cerebral haemorrhage, but haemorrhage is almost invariably fatal despite any form of treatment, while in the case of a mistaken diagnosis venesection might well cause extension of a throm-

botic lesion, it is not used nowadays for cerebral vascular lesions In some cases the primary disease, e.g. auricular fibrillation, requires treatment.

When consciousness has returned and movements of a paralysed limb begin to return, massage, graduated active exercise, and re-educative exercises will help. Active treatment should not as a rule be started until two or three weeks have elapsed Electrical treatment is popular in some quarters, but its benefit is probably only psychological Improvement may continue over a period of several months, but any effects remaining at the end of six months are almost certainly permanent After this period, massage or electrical treatment are merely a waste of time and money. Treatment now consists of persuading the patient to "make the best of his disability" In appropriate cases instructions suitable for the underlying cardiac or vascular disease should be given.

In the case of a congenital aneurysm which is causing recurring subarachnoid haemorrhage, or which has given rise to a subarachnoid haematoma, surgical treatment should be considered The treatment consists of tying the internal carotid artery In suitable cases this operation has proved highly satisfactory, but it is not devoid of danger and it may cause a hemiplegia, if indeed it does not prove immediately fatal Much depends on the exact seat of the aneurysm, and on the existence of a satisfactory collateral circulation through the vertebral artery

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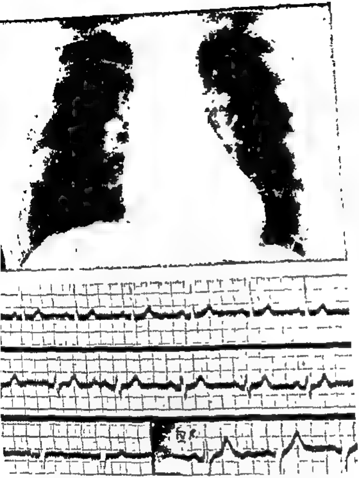

FIG 68.—Angina of effort due to rigid coronary vessels Male, 70 A month before examination he had tracheitis, a fortnight later he began to experience typical angina of effort His cardiac impulse suggested hypertrophy of the left ventricle and his heart sounds were hypertensive, the second aortic sound being "ringing", his blood pressure was 150/90 The radial and brachial arteries were not palpable and the retinal arteries were normal The cardiogram shows left axial deviation with normal R-T segments and T waves The X-ray shows a prominent left ventricular contour suggesting concentric hypertrophy despite the fact that the cardiothoracic ratio is well within the accepted normal limit (cf Fig 10), the aortic shadow is dense and a little wide, a plaque of calcification is visible in the aortic arch The clinical findings, cardiogram, and the X-ray [illegible] ... factor

CHAPTER 15

ARTERIAL DISEASE (*contd.*): CORONARY DISEASE

THE coronary arteries may be affected by arterial disease in various ways:

(1) *Sclerosis, fibrosis, or calcification of media* leads to *loss of elasticity*. There is not necessarily any narrowing. The blood supply to the myocardium remains adequate while the patient is at rest, but the coronary vessels are incapable of dilating when he exerts himself, and the blood supply becomes inadequate during effort. As long as the blood supply during rest remains adequate, no degenerative changes occur in the myocardium. The heart remains of normal size, the heart sounds remain of good tone, and the cardiogram is normal; but during effort the patient develops cardiac pain—*angina of effort*; or he may die from ventricular fibrillation if coronary vessels are unable to meet a sudden demand for enhanced blood supply.

In a case of this type, there is a genuine angina of effort *without* abnormal physical signs in the heart, and *without* abnormality in the cardiogram (though a cardiogram taken *during effort* may show temporary displacement of RT). Other vessels (radials, brachials) are usually thickened or hardened, though even this may be absent as the coronaries are sometimes affected by medial sclerosis before this becomes obvious in the vessels mentioned

(2) *Obstruction of the lumen* results from atheroma, from endarteritis, or from obstruction of the orifice by syphilitic plaques in the aorta. Obstruction may develop suddenly or gradually.

Gradual obstruction leads to ischaemia of the myocardium, which is first felt by those areas furthest removed from the smallest coronary arterioles. The myocardial fibres in these areas undergo fatty degeneration followed by necrosis and replacement fibrosis, the resulting diffuse fibrosis is described pathologically as "para-arterial fibrosis" and clinically as "chronic coronary sclerosis". The heart becomes enlarged, the first sound becomes soft, the cardiogram becomes abnormal, and the blood pressure tends to fall (especially the pulse pressure)

age of 40 is frequently (though by no means always) syphilitic, above 45 it is often secondary to atheromatous thrombosis (non-syphilitic). Coronary embolism is a rare cause. Both sexes are affected, males more often than females.

The occurrence of coronary occlusion is often the first evidence of coronary disease, the patient having previously felt perfectly well, there may have been evidence of coronary disease for a variable time previously (anything from days up to years) in the shape of angina of effort; or there may have been previous symptoms pointing to hypertension or to generalised arteriosclerosis.

Many patients on cross-examination fail to disclose any precipitating cause for the attack. A small proportion give a history of an acute infection, of some acute gastro-intestinal upset, or of some other condition which might be associated with a lowered state of general health during the few days preceding the onset of the attack. The onset is usually sudden, and it may occur at any time. In many patients it occurs during sleep, the patient goes to bed feeling in his usual health and wakens through the night with pain. When the attack develops at work, during some particular effort, or after an accident, patients are apt to blame the effort or accident and to claim compensation, it can be very difficult to come to a correct decision in such a case. According to current pathological teaching the factors which influence thrombosis are disease of the vascular endothelium, slowing of the blood stream and possibly changes in the chemical constitution of the blood. Disease of the vessel wall is present in nearly every case in the shape of atheromatous ulceration, but that alone need not cause thrombosis, and some patients wit[illegible]

[illegible] ... one accepts this view, one cannot at the same time blame effort (which speeds up the circulation) for causing the attack in another patient. It is, however, possible that some unaccustomed effort in a person with sclerotic vessels may induce spasm, and that the spasm causes slowing of the blood stream, allowing thrombosis to occur. My practice, therefore, when asked to give an opinion as to whether an attack occurring during work was precipitated by a particular

The myocardial ischaemia is felt first during effort and causes *angina of effort*—but in this case the symptom is accompanied by physical signs and cardiographic changes. As time goes on, breathlessness may take the place of pain on effort because of the increasing fibrosis of the myocardium; and ultimately the picture may be that of left or right heart failure. Less often, there is no stage of anginal pain, but insidious breathlessness on exertion progressing to established failure. The failure may be either right-sided or left-sided. As in group 1, sudden death may occur at any time, even before the stage of failure has been reached.

Sudden obstruction is usually due to thrombosis on the basis of an atheromatous ulcer; rarely it is the result of embolism, or of sudden swelling of syphilitic plaques at the orifice (e.g. a Herxheimer reaction). If the obstruction involves the main stem of a coronary artery, or one of its larger branches, there may be sudden death. If not immediately fatal, it leads to infarction of an area of myocardium corresponding to the branch affected. If the patient survives, the infarcted area is gradually replaced by fibrous tissue leaving a fibrous scar This may be small, in which case it does little damage; but if it is large it is liable to stretch, leading to aneurysm of the heart wall, with the possibility of rupture later. Rupture (with sudden death, or with haemopericardium) may also occur in the earlier stages of an infarct while it is still soft, and this is especially liable to occur if the intracardiac pressure is suddenly raised (e.g. by exercise) in the few days immediately following the occurrence of the infarct

When an infarct of the heart reaches the pericardial surface, there is a localised area of pericarditis over it. When it reaches the endocardial surface, there is a localised area of intraventricular thrombosis. This in turn may allow of portions of clot becoming dislodged to form emboli, with secondary infarction in the lungs if from the right ventricle, or in other organs if from the left ventricle.

The Symptom-complex of Coronary Occlusion

Coronary occlusion is most common above the age of 40, though it is not very infrequent in the late thirties and occasionally occurs in younger persons Coronary occlusion below the

age of 40 is frequently (though by no means always) syphilitic, above 45 it is often secondary to atheromatous thrombosis (non-syphilitic) Coronary embolism is a rare cause. Both sexes are affected, males more often than females

The occurrence of coronary occlusion is often the first evidence of coronary disease, the patient having previously felt perfectly well, there may have been evidence of coronary disease for a variable time previously (anything from days up to years) in the shape of angina of effort, or there may have been previous symptoms pointing to hypertension or to generalised arteriosclerosis.

Many patients on cross-examination fail to disclose any precipitating cause for the attack A small proportion give a history of an acute infection, of some acute gastro-intestinal upset, or of some other condition which might be associated with a lowered state of general health during the few days preceding the onset of the attack The onset is usually sudden, and it may occur at any time. In many patients it occurs during sleep, the patient goes to bed feeling in his usual health and wakens through the night with pain. When the attack develops at work, during some particular effort, or after an accident, patients are apt to blame the effort or accident and to claim compensation, it can be very difficult to come to a correct decision in such a case. According to current pathological teaching, the factors which influence thrombosis are disease of the vascular endothelium, slowing of the blood stream, and possibly changes in the chemical constitution of the blood Disease of the vessel wall is present in nearly every case in the shape of atheromatous ulceration, but that alone need not cause thrombosis, and some patients with gross atheromatous ulceration *never develop a thrombo*[illegible]

[illegible] accepts this view, one cannot at the same time blame effort (which speeds up the circulation) for causing the attack in another patient. It is, however, possible that some unaccustomed effort in a person with sclerotic vessels may induce spasm, and that the spasm causes slowing of the blood stream, allowing thrombosis to occur My practice, therefore when asked to give an opinion as to whether an attack occurring during work was precipitated by a particular

action or accident is to judge each case on its merits. If the attack occurred during some routine effort which the patient was in the habit of *making frequently without detriment*, I suggest that the development of the attack at that particular moment was purely coincidental; but if the attack occurred during some totally unaccustomed effort, while he was exposed to some unusual strain, or while he was suffering from undoubted symptoms of shock following an accident, I hold that the attack was probably precipitated by the event in question. I am not prepared to admit that an attack developing after the patient has recovered from the effects of any of the conditions mentioned (e.g. an attack occurring during the night alleged to have been precipitated by lifting a heavy weight on the previous day, or an attack occurring three or four weeks after an accident from which the patient appeared to have recovered) has any relationship to the event in question.

In 80 to 90 per cent of cases the most prominent symptom is severe pain, which may or may not be accompanied (a) by symptoms of shock, and (b) by symptoms of circulatory failure. This is often followed by a mild febrile reaction with leucocytosis lasting for two or three days.

Pain.—In a typical case the pain develops suddenly, often without warning, and becomes rapidly worse until it becomes excruciating. It usually starts behind the sternum, commonly behind the mid sternum, quite often behind the lower sternum, occasionally behind the upper sternum. It remains localised there in about half the cases. In the remainder it spreads. The radiation may be: (1) across the chest to either or both sides, more often the left than the right; (2) to either or both axillae or shoulders (more often the left), (3) down the inner side of either or both arms (more often the left), and in the arms it may be interrupted at the elbow and wrist, or may be felt there only; (4) rarely down the outer side of the arm instead of the inner side; (5) to either side of the neck, especially the left, and sometimes to the lower jaw on either side. In some cases it radiates to the back, and in others to the epigastrium. Occasionally instead of starting centrally and radiating as described, the pain starts in one of the peripheral areas mentioned and later radiates to the middle line. Exceptionally the pain is felt only at the periphery—in the arm or hand, or in the lower jaw.

In a classical case the pain lasts for a number of hours, frequently between 2 and 8, but sometimes for 24 or even 48 hours; as it subsides it leaves a dull ache which persists for a few days. However, the pain is not always continuous; sometimes it occurs in spasms or paroxysms, each lasting from 20 minutes to an hour, and separated by intervals in which there is no pain: these cases resemble a series of attacks of so-called spasmodic angina. In yet another group of cases (in which the infarct is presumably a small one) the pain is felt only on effort taking the form of abrupt onset of effort angina in a person who has previously been capable of considerable effort without any distress whatever; for example, a man who has hitherto been able to walk 10 or 15 miles, and to climb hills, etc. goes out one day and finds that after every 10 or 20 yards he develops severe pain which makes him stop; patients with this history should be diagnosed and treated as coronary occlusion not as angina of effort. Finally there are cases in which the onset is gradual, extending over several days; pain is at first mild and intermittent; during painless intervals, which may last for 24 hours or more, some patients carry on with their routine work; after a day or two pain becomes more severe and prolonged. In these cases cardiographic changes are sometimes present from the start, but sometimes they develop only after a few days.

In addition to the pain, some patients describe a sense of constriction in the chest, a feeling of choking, or a sensation of impending death. These symptoms, however, may be absent, and are not essential to the diagnosis.

Shock.—Shock is usually present if the infarct is large; with a small infarct shock may be absent, or slight and transient. Symptoms indicative of shock are pallor, faintness or syncope, dizziness, weakness, vomiting, sweating (often a "cold sweat"), coldness of the extremities, rapid pulse with small pulse volume, and collapsed veins.

Cardiac Failure.—Cardiac failure may or may not accompany the attack depending on the size of the infarct and on the state of the remainder of the myocardium. It is more often left-sided than right-sided. Some patients have severe breathlessness early in the attack, while in others it develops later; in either case signs of congestion of the lungs (rales) may follow. Sometimes there is an attack of acute pulmonary oedema

Right-sided failure is comparatively infrequent: it is shown by *distension of the neck veins, enlargement of the liver,* development of oedema, and appearance of albuminuria.

Painless Attacks.—A very small proportion of patients have *no pain during the attack, but show the remaining symptoms*—sudden onset of shock (cold sweating, etc.), left heart failure (acute breathlessness or pulmonary oedema), or right heart failure (oedema, hepatic enlargement, etc.). These were described by Gairdner as "angina sine dolore", but the name is a contradiction in terms

Physical Signs.—The findings on examination vary considerably from case to case, depending, on the one hand, on the previous state of the heart, and, on the other, on the degree of shock accompanying the attack. The classical text-book *description refers to severe attacks associated with shock.* In these circumstances patients are pale or pale and cyanosed; the skin is clammy or the victim may be in a cold sweat, he is often *restless. The pulse is rapid and feeble* The blood pressure falls and the pulse pressure is low, a normal pressure falls to 100/80, 90/75, or less, but if the pressure was previously high it may still be above normal despite a large fall, e g from 240/140 to 160/120 or 130/110 The neck veins are empty, and the veins on the dorsum of the hand remain collapsed until the hand is some distance below the level of the heart, withdrawal of blood from a vein is often difficult In contrast to these cases there are many in whom shock is slight or absent, even though pain may be severe Their appearance is normal or *flushed. The pulse rate is normal or only slightly elevated* and its volume is good. The blood pressure remains unchanged or may show an insignificant fall or slight rise, a secondary fall in pressure sometimes occurs after a few hours or a few days, in the absence of other signs of shock this is probably dependent on myocardial weakness. In a third group of patients there are signs of heart failure, breathlessness, orthopnoea, and rales in the chest indicate *left heart failure*; jugular vein distension, hepatic enlargement, and oedema point to right heart failure

The size of the heart is often normal in patients with a first attack; but if there has been pre-existing hypertension or some other cardiac lesion the heart is enlarged. The first sound becomes softer, sometimes almost inaudible. Pericardial friction can be heard in a proportion of cases at some time during

the acute stage, and is a valuable confirmatory sign; it is often transient, though occasionally persistent for several days; on occasion it comes and goes. Usually friction is localised to a small area, but in a few cases it is widespread. Gallop rhythm sometimes appears during the course of an attack which is accompanied by heart failure.

In moderate and severe cases fever appears a few hours after the onset (as shock is passing off), and for two or three days the temperature is between 99° F. and 101° F.; with this there is a leucocytosis. The milder cases remain afebrile

Cardiograms in the vast majority of cases show characteristic changes, which have been described in detail on pp. 173 to 181.

Acute Coronary Insufficiency.—Occasionally patients have an attack of severe, prolonged pain with shock and perhaps left ventricular failure, but without fever, leucocytosis, or pericardial friction In some such cases the cardiogram confirms the presence of myocardial infarction, in others the cardiographic changes merely indicate transient myocardial ischaemia without evidence of infarction. These latter cases are now termed *acute coronary insufficiency* The diagnosis is only justified after a full cardiographic exploration of the chest, but its recognition is important, as prolonged bed rest is unnecessary once the pain has subsided.

Finally in some instances occlusion and infarction develop during the course of an attack of acute coronary insufficiency; the characteristic cardiographic changes and fall in blood pressure are delayed for a few days after the onset of pain; these cases require prolonged bed rest.

Diagnosis—From *acute abdominal emergencies* such as perforation, cholecystitis, biliary colic In coronary thrombosis, rigidity is usually less marked, the fall in pulse pressure is greater, cardiographic changes are present, and the first heart sound is especially soft, pericardial friction, if present, is diagnostic Spread of pain to the arms is suggestive, but shoulder pain may occur when the diaphragmatic peritoneum is irritated

From *pericarditis*. This is often preceded by a sore throat or other symptoms of a general infection; and the onset of the cardiac symptoms is less sudden The friction is more persistent, and the cardiographic changes, if present, are different

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vious. The pain is more often localised to the cardiac apex than to the sternal region, though it may spread to the left arm.

Complications.—Cardiac failure, pulmonary oedema, and pericarditis have already been mentioned. When the infarct involves the subendocardial zone of the myocardium, a localised endocarditis is present, characterised by deposition of thrombus in the affected area, *emboli* may result, either in the lung from the right ventricle, or elsewhere from the left ventricle. Some patients have two or more *emboli*. Despite the alarm which such a course produces, recovery is possible; I have seen a patient with a coronary thrombosis, complicated by two lung infarcts within a month, make a good recovery and live for a number of years.

Rupture of the infarct is always a possibility during the first two weeks, and this may be precipitated by some sudden effort on the part of the patient, as by straining at stool or getting out of bed. Rupture is usually fatal within a few minutes, though some patients have survived with a *haemopericardium* for a few days.

In some cases, especially those with a large infarct or those who have an insufficient period of rest, the fibrous scar resulting from the infarct gradually stretches, leading to a *localised bulge*, this is described as an *aneurysm of the heart*. It is usually associated with serious limitation of the patient's cardiac reserve. As a rule there are no characteristic physical signs, the findings being merely those of a former coronary occlusion, but the condition can be recognised on X-ray examination from the presence of a localised bulge on one of the cardiac borders (Fig. 69); it may be necessary to take films at various angles other than the routine views. I have seen such an aneurysm produce a pulsating swelling on the line of the left cardiac border some distance above the apex. An aneurysm of the heart may rupture at a later date, but some patients live for a number of years—the lady in the case quoted above is known to have been alive, able to go about, and to assist in running a shop eleven years after her original attack, despite anginal attacks in the interval and a complicating diabetes.

Ventricular tachycardia is not uncommon as a complication of coronary thrombosis. The features have been described on pp. 122-3 and 126. In some cases an attack proves fatal, but in others the duration and frequency of the attacks gradually

—RT is displaced upwards in leads 1 and 2, or in all 3 leads in pericarditis.

From *pulmonary embolism.* This may cause a clinical picture indistinguishable from coronary thrombosis with central pain spreading to the arms, and a cardiogram like that of a posterior myocardial infarct. The presence of a possible source of embolism is important, also the occurrence of a blood-stained spit (not always present with embolism). Physical signs in the lungs may be diagnostic. Pulmonary embolism sometimes occurs as a complication of coronary thrombosis and may simulate a second attack.

From *simple angina.* Some cases resemble a series of attacks of spasmodic angina, others take the form of abrupt onset of effort angina. The history distinguishes the latter, the course the former, after coronary thrombosis the heart and circulation are different from what they were before the attack, but not after spasmodic angina. The differentiation from prolonged attacks of acute coronary insufficiency has been discussed above

From *intercostal herpes.* This may be difficult at first, but becomes obvious after 24-48 hours, when the rash appears.

Tabes with girdle pains might simulate coronary occlusion, but the neurological signs should prevent mistakes, where tabes and syphilitic aortitis coexist, as they sometimes do, the diagnosis may be difficult.

Root pains due to spondylitis, spinal caries, spinal metastases, or aneurysm are other possible causes of error, the description of the pain is usually quite different

Pneumonia and pleurisy are not likely to be mistaken for coronary thrombosis and can readily be distinguished by the signs in the lungs and course. *Pneumothorax* is frequently misdiagnosed as coronary occlusion, especially when on the left side Onset of pain and breathlessness during a paroxysm of coughing should raise the suspicion of pneumothorax, examination of the chest will show hyper-resonance with absent breath sounds or cavernous breathing; and X-ray will clinch the diagnosis

Neurosis may lead to attacks of pain somewhat similar to coronary thrombosis, but the other signs are absent (shock, cardiac failure, blood pressure, and cardiographic changes, etc), and other signs of hysteria or of an anxiety state are usually

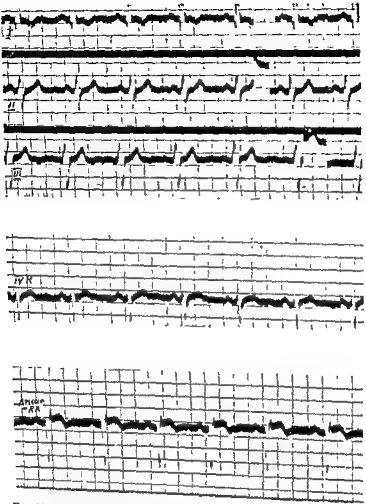

Fig 69 — Aneurysm of left ventricle following coronary thrombosis (*cont'd*).

blood sugar of 330 mg per 100 c c but no ketosis The X ray and cardio-
gram shown were obtained in February 1945 The X ray [illegible]

[illegible] small R, elevated RT, and small inverted T

Z

FIG 69 —Aneurysm of left ventricle following coronary thrombosis Female, aged 57 (February 1945), Jewish, and a diabetic of 15 years duration She was under my care in March 1937 with a severe attack of coronary thrombosis, associated with extensive pericardial friction and cardiographic changes of Q1,T1 pattern On recovery she suffered from angina of effort, BP 124/84 when convalescent in May 1937 In September 1937, five months after the initial attack, a pulsating swelling had appeared in the 4th left intercostal space on the line of the left cardiac border, blood pressure had risen to 144/96, and the X-ray picture was identical with that here portrayed. She was still alive in 1948 In the intervening eleven years angina of effort has persisted, but has gradually become less easily induced and she has done a certain amount of work running a [illegible] she says she has been feeling generally in better health [illegible], is still palpable and pulsatile, [illegible] formerly, blood pressure has risen to [illegible] by diet without insulin, but she disobeys all dietetic rules, and she has gross glycosuria with a fasting

Fig. 70.—Coronary thrombosis, anterior apical infarct causing increased translucency and lack of definition at the apex. Male, aged 62. He took an attack of sternal pain radiating to his left arm while at work two weeks before examination; the pain continued until he reached home and received pills from his doctor who described the attack as "a very severe attack of coronary thrombosis." He spent two weeks in bed, then came for examination. The cardiac impulse was impalpable, the left border 4 inches from the mid line on percussion, the heart sounds distant, and the rhythm "tic-tac." Blood pressure 129/70. Cardiogram showed inversion of T1 and T2 (indicating anterior apical infarction), in addition to left axial deviation. In the X ray film the apical region of the left ventricle is more translucent and its outline is poorly defined.

lessen in convalescence, and they may ultimately disappear. *Auricular fibrillation* sometimes appears during the acute stage of coronary occlusion; it is usually transient, occasionally persistent. *Ventricular fibrillation* is postulated as the cause of the sudden death which occurs unexpectedly in a small percentage of cases of coronary occlusion.

Course.—Many patients recover; some die during the acute stage; and some develop cardiac failure, dying after a few weeks or a few months of bedridden existence.

(1) *Recovery.* In the most favourable cases recovery is associated with good exercise-tolerance, the patient being able to lead a more or less normal life and remaining quite free from cardiac symptoms. I have known one patient who enjoyed twenty years of apparently normal health before he developed his second attack; I know of several others who have worked hard and remained symptom-free for periods up to ten years—one of them resumed his work as a miner (against advice) and continued without a day's incapacity until he was killed by a fall of coal from the roof six years later.

More often an attack of coronary thrombosis leaves behind it a greater or less degree of limitation of the cardiac reserve Frequently the limiting factor is pain, that is to say an angina of effort, but in some instances the limitation is imposed by dyspnoea. The degree of limitation is very variable, depending partly on the size of the infarct scar, partly on the state of the remainder of the myocardium, and partly on the state of the remaining coronary vessels. Many patients recover sufficiently to be able for office work or for business provided they can avoid rushing.

Patients who recover from an attack, whether they be symptom-free, limited by angina of effort, or limited by dyspnoea, sometimes develop a second or even a third attack of coronary occlusion after an interval which ranges from weeks up to years. In others, after an apparently stationary period, progressive limitation of the cardiac reserve makes its appearance and cardiac failure ensues. *Approximately one patient in ten dies suddenly and unexpectedly from ventricular fibrillation.*

(2) *Death.* During the acute stage death may occur from shock, from ventricular fibrillation, from rupture of the heart, or from embolism, or death may be gradual from progressive cardiac failure.

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(3) *Cardiac failure.* In some cases, especially those in which signs of cardiac failure have been prominent during the acute stage, the pain subsides while the symptoms and signs of failure persist. The patient may survive for a few weeks or a few months, during which he remains a bedridden or seriously incapacitated invalid.

Prognosis.—The immediate prognosis, to cover the first four weeks, depends on the degree and persistence of shock, the presence or absence of left ventricular failure, and to some extent on the age of the patient. In a series of 201 cases there were 24 deaths (12 per cent) during the first four weeks, and these all occurred in a group of 88 patients with signs of shock or cardiac failure. The mortality within a month was 48 per cent in shocked patients aged 60 or more, 29 per cent in those under 50, 19 per cent in those aged 50 to 59, and nil in patients of all ages who had no signs of shock or cardiac failure. Unfavourable prognostic signs include (1) shock which persists despite rest, warmth, and morphine, (2) a marked drop or progressive fall in blood pressure, (3) cold or cyanosed extremities, (4) a very faint or inaudible first sound, (5) gallop rhythm, (6) ventricular tachycardia.

Once the first month has been passed, prognosis is more speculative. The average duration of life is between four and five years, but the range extends from immediate death to twenty years or more. At this stage age is important, the younger the patient, the better his chances of long life. The severity of the attack has some bearing on the risk of death in the first two years, but seems to make little difference to prognosis beyond this period. Of those who survive the first month after a severe attack, 82 per cent survive for more than one year, 50 per cent for more than three years, and 24 per cent for more than five years. Approximately one-third are symptom-free, one-third have angina of effort or breathlessness but are able to work, and one-third are incapacitated.

It is a mistake to predict the ultimate working capacity until convalescence is well advanced. As a general rule, the larger the heart the greater the subsequent limitation, and incapacity is more likely in those who have had failure during the acute stage, but there are so many exceptions that any positive assertions run a grave risk of being disproved by subsequent events. Some patients with little or no cardiac

enlargement are seriously handicapped by angina of effort; others with large hearts are able to work. Predictions as to duration of life or recurrence of attacks are equally unwise.

Treatment.—Rest is of prime importance, and in order to permit adequate rest relief of pain, treatment of shock, and alleviation of respiratory distress are matters of urgency. Anti-coagulant treatment also requires consideration.

Pain. Morphine (or equivalent opium preparation) is the only drug of value for this purpose. An adult of average size should be given from ⅓ to ½ grain morphine without delay; the smaller dose may be repeated in 15 minutes if necessary. Thereafter ⅙ to ¼ grain may be given at intervals of 2 to 4 hours while severe pain persists. Amyl nitrite is generally in-effect...

formation of a large infarct.

Shock. As soon as possible the patient should be put to bed, with hot-water bottles; in the absence of respiratory distress he should be nursed flat or with the foot of the bed raised. The control of restlessness, whether due to pain or respiratory distress, by means of morphine is an essential part of the treatment of shock. Unnecessary moving of the patient should be avoided. If he has taken ill in his own home and has been put to bed he should not be moved elsewhere by ambulance until pain and shock have been relieved; if the attack has developed at work or in the street, an adequate dose of morphine should be given before removal by ambulance. "Coramine" is often given for shock in these cases but it probably makes no real difference. Shock which persists despite recumbency, warmth, and morphine involves a diminished coronary circulation has an ... of a collateral cir... infarcted area ... steps should be taken for its relief; a slow intravenous drip transfusion should be given, provided signs of cardiac failure are absent. ... in America and is th... a slight risk of a pos... excellent results fro... in this class of case given at a rate of 60 drops per minute.

Cardiac failure. When the attack is accompanied by left

ventricular failure with cardiac asthma or pulmonary oedema, the patient should be nursed in Fowler's position. Morphine will often give the desired relief. If morphine fails, theophylline-ethylene-diamine ("cardophyllin") may be tried, bearing in mind the fact that stimulation of a heart which is the seat of a soft infarct may cause rupture of the latter; nevertheless the situation is critical and the risk is justified. Digitalis should be avoided immediately after a coronary occlusion. There is less urgency in the treatment of symptoms of right ventricular failure; if these persist, digitalis or a mercurial diuretic may be used after two or three weeks, but, if given early, digitalis usually does more harm than good. Paroxysms of fibrillation, flutter, or tachycardia should be allowed to run their course; quinidine is contra-indicated until convalescence has been reached.

Anti-coagulants. It is claimed that anti-coagulants (dicoumerol or heparin) will prevent extension of the original thrombus in the coronary artery, lessen the likelihood of intraventricular thrombosis, and reduce the incidence of subsequent embolism. In the case of dicoumerol this is true only if the blood prothrombin index is reduced to less than 30 per cent, a level which involves a real risk of serious haemorrhage; any smaller reduction will prevent neither extension of the original thrombus nor subsequent embolism. Dicoumerol is justified only if facilities for daily estimation of blood prothrombin are available, vitamin K must be at hand for immediate use in case of haemorrhage. Heparin is less risky but more transient in action, frequent dosage being needed to maintain an adequate increase in the clotting time; it is an expensive form of treatment. The method of giving these drugs and controlling dosage has been described on p. 38.

Once the severe pain has been relieved, the milder ache which persists can often be controlled by "theominal" (one tablet thrice daily) or phenobarbitone (½-grain thrice daily). It is essential to ensure adequate sleep. The diet should be fluid for the first day or two; thereafter a light diet may be given for a few days, then ordinary diet. Large meals should be avoided, also bulky foods. It is best to avoid the use of purgatives during the acute stage; if necessary, an enema may be given on the third day, and gentle laxatives may be employed thereafter. Patients who recover are usually free from all symptoms

within a week; at this stage, the phenobarbitone may be discontinued, and other drugs are not required.

For the first two weeks the patient should be kept very quiet, both mentally and physically. Except for members of the family, visitors should be forbidden. He should not be allowed out of bed to stool or to the bathroom. After two weeks, if his condition is satisfactory, visitors may be allowed; and should he be one of those to whom the use of a bedpan causes difficulty and straining, he may be lifted on to a commode It is commonly taught and stated that the period of rest in bed should be six weeks, or more in cases with severe cardiac damage or with cardiac failure Personally I disagree with the fixing of a minimum limit of six weeks. It seems clear that the time required for cicatrisation of an infarct will depend on the size of the infarct; a small one will heal within three weeks, while a large one may necessitate three months There are several guides to the size of the infarct, notably the severity of the shock which accompanies the acute stage, the rapidity with which the acute symptoms pass off, the severity and duration of any pyrexia, the rapidity with which the blood pressure returns to its normal value, and the patient's general appearance and colour The size of an anterior infarct can be assessed by multiple chest lead cardiograms When all these guides point to a mild attack (although pain may have been severe at the onset) the patient may be allowed up at the end of four weeks, attacks of moderate severity require six weeks' rest, while severe attacks should be allowed eight weeks or more I have never had cause to regret the adoption of these standards

Convalescence after an attack should be gradual, the patient being allowed up for 15 minutes the first day, half an hour the next, and so on As a rough guide, the period of convalescence should be approximately equal in duration to the period of rest in bed During convalescence the patient's capacity for effort may be gauged, and he should be given instructions regarding his future life, these are discussed in connection with the treatment of angina pectoris

The Symptom-complex of Angina Pectoris

Although isolated cases of coronary occlusion had been diagnosed clinically *during the* previous century, and many

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basis unless a hypothetical coronary spasm is postulated to explain the ischaemia; and the physiologists have again altered their teaching and admit the existence of vasomotor control of the coronary vessels, so coronary spasm again becomes a possible mechanism which may be the explanation of occasional cases of spasmodic angina.

Pathological Bases of Angina Pectoris.—(1) *Coronary artery disease* is by far the most common cause. *Loss of elasticity* in fibrosis or calcification of the media will prevent vasodilatation of the coronary arteries in response to effort; myocardial ischaemia develops during effort, and ceases when termination of the effort removes the necessity for an enhanced blood supply. As already noted, there may be no abnormal physical signs on examination of the heart and no abnormality in the electrocardiogram, but radial and brachial vessels may be felt to be thick or nodular. *Narrowing of the coronary vessels* in atheroma will likewise lead to ischaemia which is relatively greater during effort than during rest, and so to angina of effort, but in this case there are degenerative changes in the myocardium producing abnormal physical signs and an abnormal electrocardiogram *Hypertension* is frequently present in cases of angina pectoris, in these cases there is *probably* disease of the coronary arteries as an underlying basis coupled with an increased load on the left ventricle due to the raised blood pressure

(2) *Aortic disease.* Angina is not uncommon in syphilitic aortitis, and is brought about by narrowing of the coronary orifices by syphilitic plaques encroaching on them When the coronary orifices are not narrowed in a case of syphilitic aortitis there is no angina Similarly, aortic atheroma does not cause angina unless the coronary vessels are also atheromatous.

(3) *Aortic regurgitation*, whether due to rheumatic carditis, to subacute bacterial endocarditis, or to syphilis, may cause angina This is because of the lowering of diastolic pressure —the coronary vessels are filled chiefly during diastole

(4) *Severe anaemias* may be associated with angina, not because of a deficiency in the quantity of blood but owing to deficiency in its quality If the blood Hbg falls to 50 per cent, double the *quantity* will be required to convey the same amount of oxygen, with 25 per cent Hbg four times the quantity will be required. The type of anaemia is immaterial

of the cardiographic features had been described by Pardee in 1920 and 1925, credit for placing the clinical diagnosis on a sound basis belongs to Parkinson and Bedford's classic paper in 1928. Previously the term angina pectoris was used to describe those cases as well as the ones now to be considered. A differentiation of sorts was sometimes made—"angina major" for the more severe attacks with more serious after effects, and "angina minor" for the less severe attacks (alternatively "angina of decubitus" and "angina of effort") —but the pathological basis of the *differentiation* was not understood, nor did the subdivision correspond accurately to the differentiation between cases with, and cases without, *coronary occlusion. Nowadays, the term "angina pectoris" is* usually applied in a more limited sense to cases in which there are attacks of myocardial pain without actual occlusion of a *vessel or infarction of the myocardium*, when the attacks are brought on by exertion and relieved by cessation of exertion, the condition is termed *angina of effort*, attacks which occur apart from effort and which are not relieved by rest are termed *spasmodic angina paroxysmal angina*, or *acute coronary insufficiency*

Mechanism.—The original theory attributed the pain in angina pectoris to spasm of the coronary arteries When it was recognised that angina could occur with disease of the aorta in persons with healthy coronary arteries, Allbutt suggested distension of the aorta due to a rise in pressure as the cause of the pain For many years discussion of the aetiology of angina pectoris took the form of a controversy between the coronary and the aortic theories When physiologists denied the existence of vasomotor nerves to the coronary vessels, the idea of coronary spasm was discredited But the recognition of myocardial infarction soon made it clear that pain can certainly arise in the myocardium, and the experiments of MacWilliam suggested that the explanation lay in continuing contractions of the heart muscle at a time when the blood supply was inadequate—the theory of "myocardial ischaemia". The vast majority of cases of angina can be explained on the basis of this theory without invoking coronary spasm or aortic distension, and most physicians now accept this view without question Very occasionally, however, a case is found which cannot be explained on the myocardial ischaemia

Symptoms.—*Angina of effort.* The characteristic feature is that the pain develops during effort; if the effort is continued, pain grows in severity and soon brings the patient to a standstill, the pain rapidly subsides (within a few minutes) when the effort ceases The situation and distribution of the pain are the same as in coronary thrombosis—sternal, or sternal and radiating; rarely radiating from periphery to sternum; and still more rarely felt in the periphery only.

There are several important points about the relation of pain to effort. Walking is especially apt to bring it on, particularly walking uphill, walking against a wind, or hurrying. Walking out of doors is more liable to cause it than walking indoors and the colder the atmosphere, the shorter the distance required to induce pain. Walking on a full stomach will bring on pain more readily than walking on an empty stomach Thus patients may say that when indoors they can go upstairs without any discomfort, but when they come to a slight rise outside they have to stop. Others say they can do quite heavy work in a factory, but cannot walk any distance outside Others have pain when going to work in the morning (full stomach after a gulped breakfast), but no pain on the way home (empty stomach) Again, some patients recognise the importance of the gastric distension factor and overlook the effort element, they say that they have pain after meals. Unless they are cross-examined and disclose that pain will only occur if they walk after a meal (not if they rest after it), the possibility of gastritis, gastric ulcer, carcinoma, etc. will be suggested, and the true diagnosis will be missed. In a given case, the amount of effort required to produce pain remains remarkably constant provided other factors are also constant (temperature, state of stomach, pace of walking, etc.). The severity of the condition is measured by the amount of effort necessary to produce pain A man who has severe pain after walking 4 miles at 4 m p h. is less seriously affected than one whose pain is only moderate but occurs on walking 50 yards at 3 m p h

Spasmodic angina. This is the name given to attacks which are induced by cold, gastric distension, or emotion in the absence of effort. Patients not infrequently develop an attack on getting into bed at night if the sheets are cold Gastric distension with gas (usually the result of aerophagy) is a

so far as causation of anginal pain is concerned. Anaemia is especially liable to cause angina if there are also minor coronary artery changes present, changes which alone would not be sufficient to do so. Or anaemia may aggravate an angina due to more advanced coronary disease. In either event improvement occurs when the anaemia is corrected.

(5) *Myxoedema* may give rise to mucinous infiltration round the small coronary twigs, and may cause angina of effort in this way—again especially if minor coronary changes are already present. Again *thyrotoxicosis* occasionally produces anginal pain by increasing the circulation rate, and hence the "load" on the myocardium, especially if early coronary changes are present.

(6) *Paroxysms of rapid heart action*, whether due to paroxysmal tachycardia, flutter, or auricular fibrillation, are sometimes (though not usually) accompanied by anginal pain. This again is more likely if there are early coronary changes present. The reason is that the increased heart rate is obtained chiefly at the expense of diastole, as shown in the appended table, and the coronary filling is therefore more likely to be inadequate.

Heart Rate	Interval between Beats	Duration of Systole	Duration of Diastole
60	1 00 second	0 40 second	0 60 second
80	0 75 ,,	0 36 ,,	0 39 ,,
100	0 60 ,,	0 32 ,,	0 28 ,,
120	0·50 ,,	0 30 ,,	0 20 ,,
150	0 40 ,,	0 30 ,,	0 10 ,,

Age and Sex.—Angina pectoris is most common in the 5th and 6th decades, i.e. the time when coronary disease has its chief incidence, occasional cases of coronary disease occur in the middle or late thirties Angina is rare below the age of 35, and is then usually due to either syphilis or to aortic regurgitation, the latter occasionally causes angina pectoris in children. Males are more often affected than females, in the proportion of about 4 to 1

Exciting Factors.—The common exciting factors are effort (angina of effort), exposure to cold, gastric distension, and emotion; or combinations of these. Attacks induced by cold, gastric distension, or emotion in the absence of effort are described as spasmodic (better "paroxysmal") angina, or *acute coronary insufficiency*.

alternatively the relief is only transient. In such circumstances morphine may be required. The associated tympanites should also receive treatment. In many cases a period of 24 hours' starvation will work wonders; the patient may be given fruit juice in small amounts at a time, sweetened with glucose.

Once the acute attack has been relieved, or when a patient is seen for the first time in an interval between attacks, it is necessary to consider first the attitude to be adopted in giving a diagnosis and prognosis. It is very important to avoid creating a superimposed anxiety state, a result which can easily be achieved if a highly strung or timid patient is told that he has angina pectoris. Lay persons have a false idea of the true state of affairs in angina; the word conjures up visions of sudden death, severe pains, and a six months' expectation of life at the outside. It is better to give the patient a simple explanation of the true nature of his pain, avoiding the label "angina"; if asked outright whether the condition is angina, it can be stated with absolute truth that "angina pectoris" simply means "pain in the chest", that there are many causes, and that some are serious, others less so. From the physician's point of view, it is important to remember that, if the patient is lucky as well as sensible and if the degree of cardiac damage is not gross, he may enjoy 10, 15, or even 20 years of relatively useful life without a great deal of discomfort; furthermore, the over-all odds against death within the first year are approximately 10-1.

General instructions applicable to all cases are as follows. The patient must avoid rushing, whether in the physical or mental sense Mentally, patients are better to be occupied than idle, but their hours must not be fatiguing, while excitement, worry, or anxiety should be avoided if possible, a philosophic outlook on life in general is of considerable help if the patient can be induced to adopt such an attitude. Physically, the amount of restriction is determined by the cardiac reserve, and must be judged individually for each case; effort should stop short of that which experience has shown him will produce pain The patients soon come to recognise the exact limitations set on them by their pain, and they should be advised to live within this limit It is permissible to use amyl nitrite or trinitrine to increase the limit, thus if a patient has to make a journey on foot, and if this journey will ordinarily

Course of Angina Pectoris.—*The course varies considerably* from case to case. In some, the condition starts insidiously, becoming steadily more severe until it culminates in death during an attack, in an attack of coronary thrombosis, or in the development of cardiac failure. In others it remains apparently stationary over a period of years. Any condition which causes a lowering of the general health may be associated with a temporary increase in the severity of the symptoms. Much that has been said regarding the course after a coronary thrombosis applies with equal force to angina pectoris; in the absence of thrombotic occlusion the average duration of life is longer, since a larger proportion of patients survive for more than four to five years.

Prognosis.—Prognosis in angina pectoris depends to a large extent on the degree of cardiac damage when the patient is first seen; the larger the heart, the more abnormal the heart sounds, and the more abnormal the cardiogram, the worse the prognosis. *Nevertheless exceptions occur, some* patients with badly damaged hearts surviving for long periods, while others with relatively minor damage succumb quickly. Other factors being equal, an angina of effort carries a less serious prognosis than a spasmodic angina, while onset in the sixties is less serious than onset in the forties. *Where some* incidental condition capable of correction is present, for example an anaemia or myxoedema, the prognosis is considerably better.

The Management and Treatment of Angina Pectoris.—The pain of an acute attack, whether it has been evoked by effort or by some other factor, is usually rapidly relieved by amyl nitrite or trinitrine. Amyl nitrite is supplied in glass capsules containing 3 or 5 minims the capsule is crushed in a handkerchief and the vapour is inhaled. Trinitrine is prescribed in tablet form, each tablet containing $\frac{1}{100}$ grain, the tablet should be chewed before being swallowed as absorption commences from the mouth Patients who take attacks should carry one or other of these drugs for use if necessary. The aim of treatment, however, should be to render the use of these drugs unnecessary, or necessary only on rare occasions, by so regulating the life and habits of the patient as to avoid attacks

Occasionally spasmodic attacks which are associated with severe gastric tympanites fail to respond to amyl nitrite, or

explanation of the true state of affairs coupled with the best prognosis which is justified, and the use of sedatives. The degree of incapacity and invalidism caused by a complicating anxiety state is often far in excess of that caused by the coronary disease. It is in these cases that the popular drug "theominal" is effective, and equally good results are obtained with phenobarbitone alone, or with ammonium bromide; the psychogenic pains are abolished, the genuine coronary pain remains unaffected.

Apart from the nitrites and the drugs used in treatment of the concomitant conditions described in the preceding paragraph, there is no evidence that drugs are of any value in angina pectoris Claims that various synthetic drugs or tissue extracts will dilate the coronary vessels are not borne out by adequately controlled therapeutic experience.

Certain surgical measures have been used in the treatment of anginal syndromes. The operation of cardio-omentopexy has been advocated in coronary thrombosis, the idea being to provide an alternative blood supply for the infarcted area of muscle, my impression is that the results, taken as a whole, compare unfavourably with those achieved by the medical measures I have recommended Two varieties of operation have been recommended in angina pectoris, namely excision of the stellate ganglion, and total thyroidectomy. Excision of the stellate ganglion has as its object the interruption of the pain tracts which convey anginal pain, and also the production of vasodilatation in the coronary circulation, it is claimed that the latter reduces the likelihood of a subsequent coronary thrombosis In some cases the operation on the left side fails to relieve the pain, and a subsequent operation on the right side is carried out There are a few cases of angina pectoris in whom benefit cannot be achieved by medical measures, and it is in them alone that I consider the operation worthy of consideration It should be preceded by alcohol injection of the ganglion, which in itself may give relief and which is less dangerous Before advising operation, the patient must be warned that, if successful, a Horner's syndrome will result One of my patients was more upset by her Horner's syndrome than by her original angina of effort The operation of thyroidectomy aims at reducing the work of the heart by diminishing the general circulation rate, it will give good

necessitate several halts, the use of a tablet of trinitrine prior to starting will often avert the development of pain, allowing the journey to be made in comfort. If the limit permits, as it does in most cases, the patient should be encouraged to continue working; or should his occupation be unsuitable, as in the case of outdoor labourers or those whose employment involves much walking and stair-climbing, they should be advised to seek a more suitable job. Similar conditions apply to games; some patients are able for golf, others are not. *It is important that the patient should have regular exercise within his limits*; insufficient exercise leads to general loss of muscle tone in which the heart shares. Dietetic habits are important. *Meals should be of small bulk; it is better* to give an extra meal a day than a large one. They should be eaten slowly, not bolted, and the patient should sit down for three-quarters of an hour after each meal. This is a factor to be considered in connection with hours of work. Any food which agrees with the patient may be allowed. "Eat what agrees with you, and avoid anything which experience has shown will cause indigestion" is the proper reply to a question regarding the nature of the diet. Sleep is also important; these patients should have 8 to 9 hours rest in bed each night, and if their sleep is disturbed, they should have a sedative. They should be warned to avoid cold as far as possible; they should remain indoors in frosty weather and on very windy days, or if they must go out they should travel by car. Their bed should be warmed before they enter it at night.

The patient's general health should be reviewed. *It is especially important to look for anaemia and treat this if present*, an improvement in the capacity for effort will follow *a rise in the haemoglobin percentage, and in some cases cure* of an anaemia will secure disappearance of anginal pain entirely, at any rate for a time. The treatment of myxoedema complicating coronary disease is discussed under "myxoedema" in Chapter 18. Obesity should also be corrected, most cases respond to restriction of salt, carbohydrate, and fat, a reduction in weight is usually associated with a corresponding improvement in the capacity for effort. In syphilitic cases treatment should start with iodides, followed by mercury and bismuth. Anxiety complicating coronary disease is treated by

CHAPTER 16

THE CARDIOVASCULAR SYSTEM IN DISEASES OF THE LUNGS, IN CHEST DEFORMITIES, AND IN DISEASES OF THE PULMONARY ARTERIES

1 The Heart in Pulmonary Embolism—Acute Cor Pulmonale

Pulmonary embolism originates from thrombus either in the systemic veins, the right auricle, or right ventricle. It therefore occurs as a sequel to venous thrombosis, especially in patients who have undergone an abdominal operation and in post-partum cases It may be a complication of auricular fibrillation or of coronary occlusion (if the latter has involved the wall of the right ventricle causing intraventricular thrombosis) Bacterial endocarditis affecting the tricuspid or pulmonic valves, a septal defect, or a patent ductus arteriosus, are other causes of pulmonary embolism, in these cases the emboli are likely to be small, and to cause relatively small infarcts

A large embolus, sufficient in size to block the main pulmonary artery, often causes sudden death, or death after a fe…

…ation of the main pulmonary artery, or in one of its main branches, produces a train of symptoms described as "acute cor pulmonale" These symptoms may also occur with smaller emboli which have merely produced a localised infarct.

An attack of acute cor pulmonale may closely resemble a coronary occlusion. The onset is abrupt with severe sternal pain and breathlessness *The pain in these cases is myocardial,* due to acute dilatation of the right side of the heart, and it may radiate in exactly the same way as the pain of coronary disease At the onset there is usually pallor which rapidly becomes accompanied by cyanosis Breathlessness is severe, often amounting to air hunger Restlessness and sweating are common Cough and spit are variable, there may be little

results if the patient's original metabolic rate is above his optimum level; this question is discussed in connection with myxoedema in Chapter 17. Like stellectomy, it is a procedure which should be reserved for patients who are severely limited by their anginal pain and who fail to improve with medical measures; such patients are few and far between. Thiouracil has been suggested as an alternative to thyroidectomy.

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1 THE HEART IN PULMONARY EMBOLISM—ACUTE COR PULMONALE

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A large embolus, sufficient in size to block the main pulmonary artery, often causes sudden death, or death after a few minutes of acute respiratory distress with pain and increasing cyanosis A rather smaller embolism which lodges at the bifurcation of the main pulmonary artery, or in one of its main branches, produces a train of symptoms described as "acute cor pulmonale" These symptoms may also occur with smaller emboli which have merely produced a localised infarct

An attack of acute cor pulmonale may closely resemble a coronary occlusion The onset is abrupt with severe sternal pain and breathlessness The pain in these cases is myocardial, due to acute dilatation of the right side of the heart, and it may radiate in exactly the same way as the pain of coronary disease At the onset there is usually pallor which rapidly becomes accompanied by cyanosis Breathlessness is severe, often amounting to air hunger Restlessness and sweating are common Cough and spit are variable, there may be little

or none, there may be coughing of blood-stained frothy mucus, or there may be a profuse haemoptysis.

On examination the patient is obviously distressed, cyanosed, or pale and cyanosed, with distended pulsating veins in the neck. The liver may be enlarged and tender. The pulse is rapid and feeble, while blood pressure drops The heart may be dilated to the right; there is marked accentuation of the second pulmonic sound, while the first sound is either soft, or short and sharp; gallop rhythm is frequent Examination of the lungs sometimes shows little abnormality: in other cases there are signs of a localised infarct (dullness, respiratory murmur either absent or tubular, and accompanied by *crepitant rale* or by *pleural friction*). The cardiogram usually shows changes similar to those of coronary occlusion with posterior myocardial infarct; Q becomes enlarged in *lead 3, RT becomes displaced upwards, and T becomes inverted.* Chest leads permit the differentiation from posterior myocardial infarction, as they show inversion of T in the right ventricular leads with prominent R, in some instances the cardiogram shows right bundle-branch block X-ray is often impracticable, but, when possible, the findings are enlargement of the pulmonary conus and right auricle. If the embolus is distal to the bifurcation of the main pulmonary artery, the hilar shadows are enlarged from congestion The development of an infarct gives rise to a localised area of consolidation in one or other lung field (Fig 72), the shadow is usually circular or irregular, occasionally wedge-shaped, it sometimes persists for several weeks, but in the case of a small infarct is often very transient disappearing within 24 to 48 hours

Treatment.—If the embolus is thought to be a large one blocking one of the main pulmonary artery branches, and if surgical aid is immediately available, the operation of embolectomy offers the best chance of survival, slender though this may be. If the affected branch is smaller (as shown by the presence of clinical signs of a localised infarct), so that the embolus is unlikely to be removable surgically, morphine should be given immediately in full dosage (a quarter or even a third of a grain); a ¼-grain dose may be repeated in 15 minutes if it has not acted. Venous distension in these cases is thought to be helpful; although the cardiac output is reduced, the patient has to depend on his high venous pressure for main-

Fig. 72.—Lung infarct. Female aged 48 with mitral stenosis, aortic regurgitation, and auricular fibrillation. She had a previous lung infarct a year before. Fourteen days prior to the date of this X-ray she had two emboli, one in the left anterior tibial artery and one in the right lung. The X ray shows filling in of the left middle arc due to enlargement of pulmonary artery and pulmonary conus, considerable enlargement of the left ventricle, and enlargement of the right auricle. The shadow of the lung infarct is seen in the right mid zone, and a small effusion is present at the base of the right pleural cavity. The right hilar shadow is somewhat enlarged, but the remainder of the lung fields is remarkably free from signs of congestion. The patient made a good recovery from both her emboli and was discharged from hospital after 6 weeks under digitalis control; the circulation in the left foot was good and the signs in the right lung had disappeared. Another example of lung infarct is shown in Fig. 66, p. 293.

tenance of what little output he can squeeze past the embolus (McMichaël, 1946). In these circumstances venesection is contra-indicated and digitalis is more likely to do harm than good. The pleuritic pain of a localised lung infarct is occasionally eased by heat (kaolin poultice), but if this is ineffective, morphine should be used. Once the acute distress has been relieved, subsequent treatment is a matter of rest and nursing for four to six weeks, depending on the severity of the case. The source of embolism should also be considered. In cases of venous thrombosis, the use of dicoumarin may avert further thrombosis by lengthening the coagulation time, this drug is well worth trying in cases of recurring pulmonary embolism secondary to venous thrombosis. Bacterial endocarditis affecting a patent ductus arteriosus calls for ligation of the duct. When embolism has been secondary to auricular fibrillation or to coronary thrombosis, these are treated in the usual way

2. The Heart in Pneumothorax

The sudden development of a pneumothorax sometimes produces effects on the circulation similar to those caused by a pulmonary embolism. Collapse of the affected lung greatly reduces the vascular bed with results analogous to those of an embolus blocking one of the main branches of the pulmonary artery Alternatively, the pain and pulmonary collapse which follow rupture of the pleura often produce reflex shock ("pleural shock") Finally, in cases of valvular pneumothorax the rise in intra-pleural pressure may be sufficient to cause mediastinal displacement with mechanical embarrassment of the circulation.

The onset is abrupt, often during a bout of coughing. While the pain in many cases is localised to the affected side of the chest, there are instances in which it is myocardial in origin and in distribution. Such cases are not infrequently diagnosed as coronary occlusion, the pneumothorax being overlooked With the pain there is pallor or pale cyanosis, breathlessness, rapid pulse, fall in pulse pressure, and sometimes sweating, the neck veins may be collapsed or distended, depending on whether reflex shock or cardiac embarrassment predominates. The cardiogram is often normal, but in some cases it resembles that of pulmonary embolism. The pulmonary signs are diminished movement and hyperresonance, vocal fremitus and respiratory

murmur are absent with a closed pneumothorax; an open pneumothorax gives increased fremitus and amphoric breathing. A valvular pneumothorax is characterised by increasing respiratory distress, increasing venous distension, and progressive displacement of the heart. The pulmonary signs distinguish the acute cor pulmonale of pneumothorax from that of pulmonary embolism, absence of a source of embolism and onset during a bout of coughing are other distinguishing features The X-ray appearances are characteristic.

The initial treatment is that of shock, viz. rest, warmth, and morphine In most cases the shock passes off and any cardiac embarrassment subsides as the lung re-expands. In cases of valvular pneumothorax with increasing respiratory distress, a needle should be inserted into the pleura and air should be allowed to escape slowly, the pressure should be reduced gradually Digitalis is of no value and may be dangerous, as in pulmonary embolism

3. The Heart in Emphysema—Chronic Cor Pulmonale

Breathlessness on exertion is the most constant symptom of emphysema In the earlier stages the heart is still normal, and the breathlessness is pulmonary in origin Two factors participate in its causation, namely lowered vital capacity resulting from the diminished chest expansion, and loss of respiratory epithelium consequent on disruption of many of the inter-alveolar septa. Even in these mild cases in which the heart is still normal, emphysema produces certain physical signs which might (and sometimes *do*) lead to an incorrect diagnosis of heart disease, owing to the interposition of emphysematous lung between the cardiac apex and the chest wall, the apex impulse becomes feeble or impalpable, and the sounds are distant

In more advanced stages of emphysema, pulmonary hypertension develops and is followed by hypertrophy of the right ventricle and sometimes right auricle The arterial oxygen content is diminished and the circulation rate is increased in consequence Congestive failure may ensue, usually gradual but occasionally sudden in onset In the "compensated" stage it is usually impossible to demonstrate the cardiac enlargement clinically, and as a rule there are no characteristic

clinical signs; sometimes the pulmonic second sound is accentuated; the degree of breathlessness and cyanosis no doubt increase, but there is no clinical means of differentiating

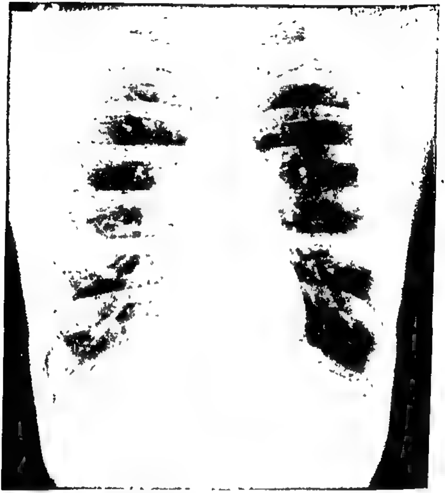


FIG. 73.—Teleradiogram showing early cardiac involvement in emphysema, from a long-standing case of asthma, female, aged 33. The heart shadow as a whole is small, but there is decided prominence of the conus pulmonalis. (By courtesy of Dr D M Harper.)

the extent to which they are pulmonary or cardiac respectively. Estimation of the cardiac output demonstrates that the circulation rate is increased, often considerably. X-ray is the most valuable diagnostic procedure at this stage; enlargement of the conus pulmonalis, main pulmonary artery branches, and right ventricle can be demonstrated; less often the right

FIG 74.—Advanced cor pulmonale secondary to chronic pulmonary fibrosis and emphysema, and complicated by hypertension Male, aged 51, a sheet-metal worker History of bronchitis and asthma for many years,

present Cardiac apex 4 inches from mid line, right border at right sternal edge, sounds of fairly good tone, aortic second accentuated Chest clinically has the shape of advanced emphysema and shows impaired resonance with crepitation at the left base Copious albuminuria present but no red blood cells or casts Cardiogram shows a low R and deep S in all leads, S2 rather broad Note the marked prominence of conus pulmonalis and pulmonary artery, aortic knob dense and somewhat prominent, left ventricular contour prominent. There is pulmonary emphysema with patchy fibrosis (? siderosis), and calcification of the pleura in the left lower zone

auricle is also enlarged. In the electrocardiogram, P may become low in lead 1 and prominent in lead 3, suggesting right auricular hypertrophy. The ventricular complexes may show right axial deviation; but normal axis or left axial deviation are each equally frequent owing, on the one hand, to the altered shape of the chest (which may produce a transverse lie of the heart), and on the other to associated hypertension with left ventricular enlargement. Many cases of emphysema

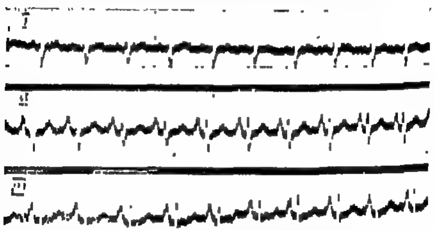


FIG. 75 – Cardiogram in cor pulmonale. From a case of emphysema. The P waves are low in lead 1, large and pointed in leads 2 and 3, being larger in lead 3 than in lead 2. There is right axial deviation.

are complicated by hypertension or by arteriosclerosis which produce left ventricular enlargement and modify the physical signs accordingly.

With the onset of failure, cyanosis increases and breathlessness becomes more severe; it may become worse in paroxysms associated with bronchospasm. The neck veins become distended, the liver enlarges, while oedema and albuminuria complete the picture. Signs of right ventricular hypertrophy (p. 166) appear in the cardiogram. The rhythm usually remains normal even with failure; auricular fibrillation is rare. Until the onset of failure, the heart rate is rarely increased. Hypertension not infrequently develops during right heart failure due to emphysema, and it may disappear when the patient improves (see legend to Fig. 74); the mechanism is obscure.

Other chronic lung diseases, such as silicosis, pulmonary fibrosis, etc., can produce similar effects on the heart and circulation (Fig. 74)

Treatment in the earlier stages is a matter of attempting to improve the vital capacity by appropriate (expiratory) breathing exercises. An effort should be made to delay the onset of the later stages by ensuring that the patient's occupation and habits are suitable, heavy manual labour, and occupations involving attempts to expire against a resistance (e g glass-blowing, wind-instrument playing), are unsuitable. Intercurrent colds or bronchitis should be avoided if possible by abstention from ill-ventilated and overcrowded places; and those who can afford the change should seek an equable climate during the winter months.

Once the stage of failure is reached, treatment is by rest in bed with oxygen for cyanosis, mercurial diuretics for oedema, and sedatives for relief of distress. The value of digitalis in these cases has been questioned by McMichael in view of the high cardiac output he contends that, far from increasing the output, digitalis is likely to lower it by reducing the venous pressure, and he claims to have seen an injection of digoxin followed by a substantial fall in cardiac output and by death. Clearly, similar reasoning is applicable in any case of right heart failure in which the summit of Starling's curve has not been reached (see Chapter 1, p 2), but this is not a valid argument against the use of the drug in cases in which the summit has been surpassed, whether the circulation be hyperkinetic, orthokinetic, or hypokinetic. Clinical therapeutic experience indicates that digitalis is valuable in many cases of emphysema heart failure though not in all, and most clinicians continue to prescribe the drug. As there is no clinical means of assessing the point on Starling's curve which has been reached by the patient, it is probably wise to avoid massive oral or intravenous dosage, and to use the routine four-hourly dosage advocated in Chapter 23, p 453 Analogous considerations apply to venesection

4 The Heart in Chest Deformities

The effects of chest deformities on the heart may be considered from two entirely distinct aspects In the first place,

auricle is also enlarged. In the electrocardiogram, P may become low in lead 1 and prominent in lead 3, suggesting right auricular hypertrophy. The ventricular complexes may show right axial deviation; but normal axis or left axial deviation are each equally frequent owing, on the one hand, to the altered shape of the chest (which may produce a transverse lie of the heart), and on the other to associated hypertension with left ventricular enlargement. Many cases of emphysema

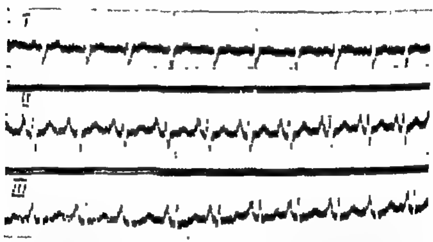
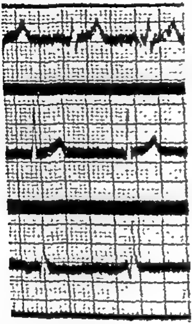


Fig. 75.—Cardiogram in cor pulmonale From a case of emphysema. The P waves are low in lead 1, large and pointed in leads 2 and 3, being larger in lead 3 than in lead 2. There is right axial deviation.

are complicated by hypertension or by arteriosclerosis which produce left ventricular enlargement and modify the physical signs accordingly.

With the onset of failure, cyanosis increases and breathlessness becomes more severe, it may become worse in paroxysms associated with bronchospasm. The neck veins become distended, the liver enlarges, while oedema and albuminuria complete the picture Signs of right ventricular hypertrophy (p 166) appear in the cardiogram The rhythm usually remains normal even with failure, auricular fibrillation is rare Until the onset of failure, the heart rate is rarely increased. Hypertension not infrequently develops during right heart failure due to emphysema, and it may disappear when the patient improves (see legend to Fig. 74), the mechanism is obscure

one side and flatter than usual on the other; there is a "hump" on one side of the sternum with a more or less flat backward slope on the other. Even these comparatively mild grades of scoliosis are sufficient to cause displacement of the cardiac impulse in relation to the mid line of the sternum, and the



76

FIG. 76.—Distortion of heart shadow due to scoliosis. Male, aged [illegible]

[illegible]

[illegible] certainly due solely to the scoliotic deformity. The cardiogram showed slight *right* axial deviation and inversion of T_3. This is a dubious case, requiring further observation before any definite opinion as to the presence or absence of carditis can be expressed. It illustrates well the degree of scoliosis which can sometimes remain unsuspected clinically, and the difficulty attending diagnosis in such cases.

minor deformities of the chest may cause displacement of the heart, thereby producing abnormal physical signs which might lead to errors in diagnosis. In the second place, severe chest deformities may in themselves cause circulatory failure.

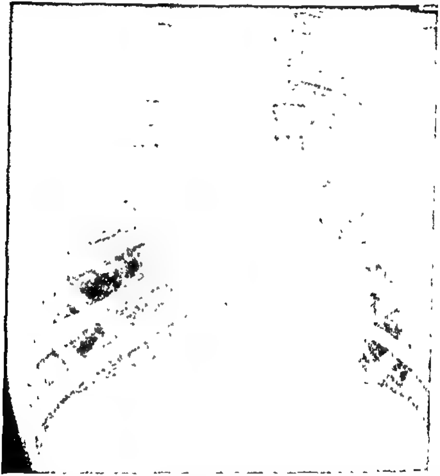


Fig. 76

Alterations in the Normal Physical Signs produced by Minor Chest Deformities.—*Scoliosis* is a relatively common condition. Its degree as shown radiologically is often considerably greater than that expected clinically from inspection of the back, and there are some patients in whom scoliosis is well marked radiologically though it has remained unsuspected from clinical inspection of the back. These cases, however, can usually be suspected from clinical examination of the front of the chest, for the anterior po ions of the ribs are more prominent on

e had pain in the left side of her chest, worse on coughing; e worked until 9th February but collapsed on going to visit r doctor on 12th February. I saw her four days later. Her in was pale, malar regions slightly flushed, lips and cheeks anosed; breathing was laboured, respiration rate 38 per inute, pulse rate 96, and temperature normal. There were gns of consolidation with crepitant rale in both lower lobes, specially the left. Cardiac dullness was enlarged to the right ut not to the left, the apex being 3½ inches from the mid line, he sounds were of quite good tone, blood pressure, 174/126. There was no enlargement of the liver and no oedema. Clinic-lly there seemed no doubt that she had a bronchopneumonia, robably non-specific but possibly tuberculous, this diagnosis ppeared to be confirmed by the X-ray taken on 23rd February Fig 77), although the fact that her illness was afebrile through-out raised some misgivings The cardiogram (Fig. 77) was that of chronic cor pulmonale. There was improvement in her respiration and cyanosis until 1st March, when she relapsed, becoming distressed, breathless, and cyanosed, she died a few hours later.

Post-mortem examination (Dr. W. B Davis) showed small lungs, both lower lobes appearing somewhat collapsed, there was no sign of consolidation or of tuberculosis, there was no gross oedema, but a marked degree of congestion. A careful histological search failed to show any inflammatory lesion (apart from old pleural thickening at the left base), and the bronchioles were described as "remarkably free from infection". The heart weighed 22 oz., there was gross hypertrophy and dilatation of the right ventricle, the myocardium being three to four times its normal thickness, the myocardium of the left ventricle was of normal thickness, there was no valvular lesion, pericardial disease, septal defect, or coronary disease. Clearly the intense pulmonary congestion in this case (sufficient to simulate bronchopneumonia both clinically and radiologically) cannot be attributed to left ventricular failure, it can only be explained on the basis of primary stasis in the lungs resulting from the mechanical interference with the normal respiratory movements

A second mechanism which can impair ...
efficien...

apex will often be found in the nipple line or beyond (4 to 4½ inches from the mid line) even though there be no real cardiac enlargement. In the presence of such a deformity of the chest wall, enlargement of the heart should never be diagnosed until it has been confirmed radiologically; it will often be found that a heart which has seemed to show considerable enlargement clinically is, in fact, of normal size or even smaller than average. Another comparatively common deformity which leads to displacement of the heart is depression of the lower end of the sternum; the costal cartilages slope inwards to form a trough at the base of which the sternum lies; in some cases the sternum is so closely approximated to the anterior surfaces of the vertebral bodies that there is no room for the heart between them, and the whole heart is bodily squeezed to the left. The right border of the heart lies to the left of the sternum, and the apex lies somewhere between the nipple and the anterior axillary line. In these cases, too, X-ray examination is essential before any opinion can be expressed regarding the size of the heart. Patients in whom the heart and mediastinum are displaced by either of these deformities not infrequently have systolic murmurs at the base of the heart, and the second sound at the pulmonic area is often relatively accentuated.

Effect of Chest Deformities on the Efficiency of the Heart and Circulation.—Severe chest deformities militate against an efficient circulation in several ways. One of the most important is in consequence of the serious restriction in the respiratory movements; the circulation thereby loses the valuable aid which the respiratory movements provide in assisting the venous return, a loss which can lead, on the one hand, to congestion of the veins outside the thorax, and on the other to a diminished cardiac output. Furthermore, interference with the movement of expiration abolishes the assistance given in the emptying of the lung capillaries and veins; hence pulmonary congestion may arise. This mechanism is well illustrated by the following case.

A small woman, aged 41, had an extreme dorsal kyphoscoliosis. During adolescence she had an attack of left-sided pleurisy. She subsequently worked as a clerkess. From time to time she complained of dyspeptic symptoms which were attributed to a duodenal ulcer. In November 1944 she developed a cough and spit; towards the end of January 1945

she had pain in the left side of her chest, worse on coughing; she worked until 9th February but collapsed on going to visit her doctor on 12th February. I saw her four days later. Her skin was pale, malar regions slightly flushed, lips and cheeks cyanosed; breathing was laboured, respiration rate 58 per minute, pulse rate 96, and temperature normal. There were signs of consolidation with crepitant rale in both lower lobes, especially the left. Cardiac dullness was enlarged to the right but not to the left, the apex being 3½ inches from the mid line; the sounds were of quite good tone; blood pressure, 174/126. There was no enlargement of the liver and no oedema. Clinically there seemed no doubt that she had a bronchopneumonia, probably non-specific but possibly tuberculous; this diagnosis appeared to be ...

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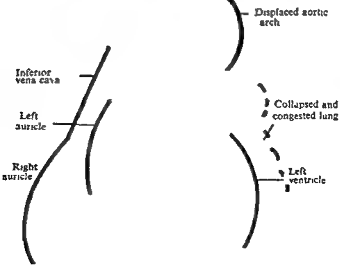

Displaced aortic arch
Inferior vena cava
Left auricle
Right auricle
Collapsed and congested lung
Left ventricle

77

severe chest deformities are apt to have partial collapse in some areas of the lung with compensatory emphysema in others; and they are prone to bronchitis; all of which combine to produce an effect on the heart similar to that of emphysema.

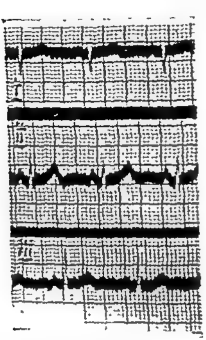

FIG 77—Extreme kyphoscoliotic deformity of chest, gross right sided cardiac enlargement, intense pulmonary congestion simulating bronchopneumonia (For case description see text)

Clinically, the manifestations commence with breathlessness and increasing cyanosis There is often a persistent tachycardia which may represent a foredoomed attempt to compensate for the d...

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appear. In these cases breathlessness is sometimes worse when *the patient is propped up than when lying flat*; *and it may be* worse when lying on one side than on the other. The cardiographic findings are similar to those in emphysema. Treatment can only be symptomatic. Morphine is contra-indicated in these cases; several examples of collapse and death following a first injection of morphine have recently been recorded, and the patient described above had her only injection of morphine a few hours before her death. *Oxygen is indicated. The use* of digitalis or of venesection is governed by the same considerations as in emphysematous heart failure (p. 355).

5. Pulmonary Artery Disease

The syndrome associated with disease of the pulmonary vessels is now generally known as "pulmonary hypertension". It is usually secondary to mitral stenosis or to emphysema, either of which causes a rise in the pulmonary arterial pressure. The atheroma usually found in the pulmonary arteries in these cases is regarded as secondary to the raised pressure. Dilatation of the pulmonary artery also occurs as a sequel to congenital lesions which involve a left to right shunt, viz. in the late stages of atrial septal defect (Fig. 82, p. 405), in Eisenmenger's complex, and in patency of the ductus arteriosus (Fig. 85, p. 413), the pulmonary arterial pressure rises in consequence of these lesions, and atheroma may follow. Syphilis occasionally affects the pulmonary arteries. Thrombosis may occur as a sequel to atheroma or to syphilis, it has also been described in sickle-cell anaemia.

Ayerza described certain cases which he attributed to primary sclerosis of the pulmonary artery (Ayerza's disease); doubt has since been cast on his claims, the suggestion being that some of his cases were atheroma secondary to emphysema, others being syphilitic. I have, however, seen one case of Dr. Ivy Mackenzie's which seems to correspond to a primary sclerosis of the pulmonary arterial tree, there was neither mitral stenosis, emphysema, nor a congenital lesion, and the Wassermann was negative; the systemic arteries were unaffected, the lesions being confined to the pulmonary vessels. The latter showed diffuse hyperplastic changes resembling those seen in the peripheral vessels in hypertension, in places there

was thrombosis and in others canalisation of organised thrombi reminiscent of the appearances in thrombo-angiitis obliterans. Recently certain cases of "primary pulmonary hypertension" have been described in which no cause could be demonstrated; the pulmonary arteries showed secondary atheroma but no primary sclerosis.

The symptoms of pulmonary hypertension, whether secondary or primary, are breathlessness and cyanosis followed by right ventricular failure. Cyanosis is often extreme and may suggest that of congenital heart disease. The physical, radiological, and electrocardiographic findings are those of right ventricular hypertrophy and right ventricular failure. In cases associated with thrombosis, haemoptysis is a prominent symptom sometimes recurrent, sometimes persistent. The diagnosis of primary pulmonary hypertension depends on the exclusion of congenital lesions and of conditions which may cause secondary pulmonary hypertension. The prognosis is bad. Treatment is purely palliative, and is that of right ventricular failure, as in emphysema.

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CHAPTER 17

THE CARDIOVASCULAR SYSTEM IN ENDOCRINE DISORDERS, DEFICIENCY DISEASES, AND ANAEMIAS

1. The Heart in Thyrotoxicosis

The earlier cardiac and circulatory manifestations of thyrotoxicosis suggest over-stimulation of a healthy circulatory mechanism. The increased basal metabolism of thyrotoxicosis involves an augmented circulation rate and a greater cardiac output; part of the circulatory reserve is already being utilised at rest, leaving a smaller margin to meet the demands of exercise. In prolonged or more severe cases, cardiac hypertrophy follows; auricular fibrillation is a very frequent complication; cardiac failure ultimately ensues. The cardiac output at the onset of thyrotoxic heart failure is still above normal, though insufficient to supply the increased requirements of the body. The older the patient the greater the likelihood of cardiac hypertrophy of auricular fibrillation, and of cardiac failure. Thyrotoxicosis developing below the age of 30, that is to say when the circulatory organs are in their prime, tends to exert its most deleterious effects on the central nervous system; auricular fibrillation and congestive failure are rare ("primary toxic goitre"). Thyrotoxicosis developing after the age of 40, that is to say in persons whose circulatory organs are beginning to age, produces auricular fibrillation and cardiac failure very frequently ("secondary toxic goitre", "toxic adenoma"). My personal belief is that the distinction between so-called primary and secondary toxic goitre with regard to their relative effects on the nervous and circulatory systems is due to the difference in their age incidence and not to any fundamental difference in their nature.

Pathologically, hypertrophy of the left ventricle is commonly found (though in many cases the heart is small during life); the auricles may be dilated (auricular fibrillation) and may contain thrombus. Histologically, areas of degeneration, areas of lymphocytic infiltration, and small fibrous scars are

sometimes found, according to Fahr (quoted by Aschoff); but in the majority of cases there are no characteristic histological findings Signs of congestive failure, or infarcts which have resulted from emboli, may be present.

In the first stage the most frequent cardiac symptom is palpitation felt on excitement or on exertion. At this stage breathlessness is usually absent. Examination reveals a persistent tachycardia, the pulse is regular, its rate may be anywhere between 100 and 140 (or even more), and the sleeping pulse rate is also elevated, the pulse volume is large. Exercise causes a larger increase than in health, e g. to 160 or 170, but the absence of breathlessness with this is often striking, the return to the resting value may be rapid (within 30 to 60 seconds) or delayed The systolic blood pressure is elevated the diastolic normal or low, so that the pulse pressure is much increased The cardiac impulse is diffuse, excited, and rapid but the apex is not displaced, in fact X ray examination will usually show that the heart is normal or even below average in size, the pulmonary conus may be more prominent than usual. The heart sounds are loud the first sound frequently intoned and the second sound often accentuated at the pulmonic area; a soft systolic murmur is heard in many cases either at the apex or at the base—the innocent systolic murmur of tachycardia Cardiograms show a simple tachycardia, the P and T waves are often relatively high, and the PR interval is comparatively short Signs of venous congestion are absent, on the other hand signs of vaso-motor instability are frequent, the most constant of them being tache (dermatographism)

The differential diagnosis at this stage is from anxiety neurosis, which may give rise to similar cardiovascular manifestations Difficulty arises chiefly in those cases in which the cardiovascular features are prominent while the remaining signs of thyrotoxicosis are slight, a type of case which is by no means uncommon A history of loss of weight, presence of thyroid enlargement, presence of a systolic murmur or thrill over the superior *thyroid* arteries or presence of eye signs favour a

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stage is that of the thyrotoxicosis Rest

and sedatives

bromide) are required by all but the mildest cases. Thiouracil in doses of 50 to 200 milligrams thrice daily will reduce the basal metabolic rate and control the circulatory manifestations of thyrotoxicosis in the majority of cases, especially in the younger age groups; in some, a prolonged course of thiouracil will effect a cure. In older patients and in those with adenomatous goitres it sometimes fails to give satisfactory control, and in these circumstances thyroidectomy should be advised without awaiting the development of cardiac hypertrophy, auricular fibrillation, or cardiac failure. Frequent leucocyte counts are essential during thiouracil treatment The important point from the cardiological standpoint is that the cardiovascular manifestations respond to treatment of the primary condition, and to that alone Digitalis is contra-indicated; it is never of benefit at this stage in the disease and it may do considerable harm.

Not infrequently it happens that thyrotoxicosis develops in the subject of an old-standing rheumatic valvular lesion usually mitral stenosis. The physical signs are correspondingly modified, the characteristic murmur or murmurs being present. Provided the case conforms to the description given above, and that cardiac failure has not developed, treatment is that described for uncomplicated thyrotoxicosis Similar considerations apply when thyrotoxicosis complicates hypertension.

In the second stage evidence of cardiac hypertrophy or of circulatory insufficiency is present in addition to the foregoing findings. The patient complains of breathlessness on exertion as well as of palpitation, and there is often a history of slight oedema of the ankles. The pulse pressure may be lower. The cardiac apex is displaced to the left, and X-ray shows some degree of cardiac enlargement. The P and T waves in the cardiogram are lower; and in some cases there is evidence of myocarditis in the shape of delayed conduction or of slight displacement of the RT interval

Rest and sedatives are imperative in these cases Usually any oedema disappears with this treatment, and digitalis is unnecessary, but if oedema persists, it may be required Unless there are signs of considerable improvement within a few days on rest and sedatives, a course of iodine should be given. If, at the end of 14 days on iodine, the patient's cardiac and general condition will permit of operation, this should be

carried out without further delay. It may be, however, that the patient's condition will not permit of operation, in which case the iodine should be discontinued; after a few weeks' further rest and general treatment a second course of iodine will probably render the patient fit for operation. Many physicians use thiouracil in this group of cases also; my personal experience is that fewer cases are satisfactorily controlled at this stage. Operation provides a more speedy cure; it eliminates the danger of further cardiac damage arising from delay in controlling the thyrotoxicosis, and, in the hands of a competent surgeon, it involves no greater risk than thiouracil.

The most severe grade of cardiovascular damage is presented by those cases with auricular fibrillation which is soon followed by symptoms of congestive failure. In the majority of these cases the heart is enlarged, sometimes considerably so, although I have seen cases with auricular fibrillation and a heart of normal size. Signs of congestive failure are usually present, occasionally absent. In addition to rest and sedatives, digitalis is indicated when signs of congestive failure are present. In some cases digitalis is ineffective until iodine is given in addition. Operation should be undertaken once the signs of congestive failure have cleared up and the heart rate has been adequately controlled. It is better to operate at that point than to delay in the hope of restoring the normal rhythm with quinidine; indeed it is usually a waste of time to attempt quinidine therapy for the fibrillation until the thyrotoxicosis itself has been dealt with, and in my experience the operation is no more dangerous with a fibrillating heart than with normal rhythm; the majority of the few operative deaths I have seen have occurred in patients with normal rhythm. After operation, an interval of at least a month should be allowed for spontaneous restoration of normal rhythm. If at the end of that time the heart is still fibrillating, and if there are no contra-indications to the use of quinidine (see pp. 139 and 140), a course should be given. Some of the most gratifying results in cardiology are obtained in these cases.

2. The Heart in Myxoedema

Some patients with myxoedema are free from cardiovascular symptoms, apart from a tendency for the pulse ...

slow and the blood pressure low, the cardiovascular system is entirely normal A considerable number of patients with myxoedema do exhibit cardiovascular symptoms and signs; these may be due to independent cardiovascular disease, often

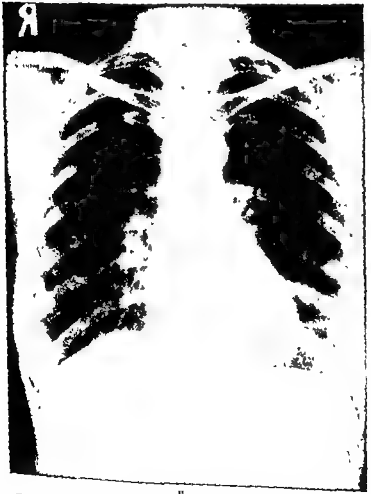
A

Fig 78a —Generalised enlargement of the heart in myxoedema—uncomplicated myxoedema heart (Dr A M Scott's case)

A Before treatment

arteriosclerosis, coronary disease, or hypertension; but there is a group of cases in which there are cardiovascular manifestations peculiar to myxoedema and curable by thyroid therapy; these are known as " myxoedema heart ".

Myxoedema Heart.—The pathological basis of "myxoedema heart" is still undecided. The gross enlargement of the heart has been attributed on the one hand to pericardial effusion,



B

Fig 78B —Generalised enlargement of the heart in myxoedema (*contd*)

B After eight weeks treatment with thyroid

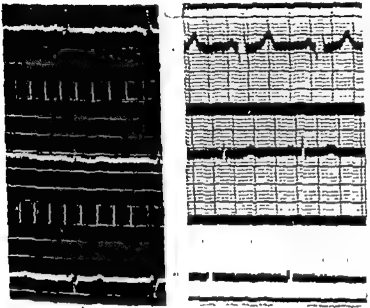
and on the other to mucinous (myxoedematous) infiltration of the myocardium, it differs from all other forms of cardiac enlargement (pericardial effusion excepted) in that it will disappear completely under thyroid therapy (Fig 78) The

symptoms have variously been attributed to mucinous infiltration of the myocardium, to the effects of hypoglycaemia, and to the anaemia which so frequently complicates myxoedema and which might well be associated with fatty changes in the heart muscle.

The clinical features were first described by Zondek in 1918. Breathlessness on exertion is a common early symptom, and it may be followed by oedema of cardiac type with all the signs and symptoms of congestive failure. Slight precordial aching is another common complaint. Some patients describe a continuous dull ache made temporarily worse by exertion; while others have a typical angina of effort, or attacks resembling spasmodic angina. Sudden attacks of collapse with pallor, cold sweating, and perhaps a feeling of oppression in the chest are occasionally met with, and one of my patients was found unconscious after such an attack, which seems to have been the starting-point of her illness. The physical signs are: *a feeble or impalpable cardiac impulse*; *distant heart sounds*; a small, slow pulse with a low blood pressure; and cardiac enlargement of varying degree With these, there may be signs of congestive failure. *X*-ray examination shows the cardiac enlargement to be generalised, and sometimes considerable in degree; the normal heart shape is usually preserved, though in some cases the shape suggests a pericardial effusion; on screening, the cardiac pulsations are seen to be of small extent. The cardiogram shows flattening of the P and T waves, which may become so small as to be indistinguishable; the QRS complexes generally remain normal, the heart rate is slow, and there may be lengthening of the PR interval up to about 0·24 second, though in many cases the conduction time remains unaffected. T is sometimes inverted (as well as shallow) in lead 1, or in leads 1 and 2, this finding probably implies independent coronary disease (see Figs. 79 and 80).

In rare instances the signs and symptoms of myxoedema heart are found in patients who present neither the appearance nor the other clinical manifestations of myxoedema On investigation, these patients show a low basal metabolic rate and a raised blood cholesterol. The symptoms are relieved and the cardiac enlargement disappears on treatment with thyroid. The condition has been named "Abortive myxoedema" or "Abortive myxoedema heart" by Zondek (1941)

The treatment of myxoedema heart consists of rest coupled with administration of thyroid extract. The initial dose of the latter should be small, and it should be increased at intervals of a few days until the optimum dose for the patient in question is found. At the same time any coexisting anaemia should be



A B

FIG. 79.—Cardiogram in myxoedema.

A *Female* [illegible]

B *Same patient, October 1932.* Has been on thyroid grs. 3 daily, ever since. No further anginal pain. BMR –15%. No clinical signs of myxoedema. The voltage of all deflections has increased, but especially the P and T waves. T1 is large and upright, T3 shallow and inverted.

treated, blood examination will determine whether iron or liver is indicated. Attacks of collapse and spasmodic attacks of anginal pain can often be prevented by giving glucose. Under the influence of thyroid, congestive symptoms gradually clear up, and the heart becomes steadily reduced in size, as can be demonstrated by serial X-ray examinations, in due course the enlargement disappears entirely. It should be noted that these cases do not respond to the usual treatment for congestive failure, namely rest and digitalis, the latter drug

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The clinical features were first described by Zondek in 1918. Breathlessness on exertion is a common early symptom, and it may be followed by oedema of cardiac type with all the signs and symptoms of congestive failure. Slight precordial aching is another common complaint. Some patients describe a continuous dull ache made temporarily worse by exertion; while others have a typical angina of effort, or attacks resembling spasmodic angina. Sudden attacks of collapse with pallor, cold sweating, and perhaps a feeling of oppression in the chest are occasionally met with, and one of my patients was found unconscious after such an attack, which seems to have been the starting-point of her illness. The physical signs are: a feeble or impalpable cardiac impulse; distant heart sounds, a small, slow pulse with a low blood pressure; and cardiac enlargement of varying degree. With these, there may be signs of congestive failure X-ray examination shows the cardiac enlargement to be generalised, and sometimes considerable in degree; the normal heart shape is usually preserved, though in some cases the shape suggests a pericardial effusion; on screening, the cardiac pulsations are seen to be of small extent. The cardiogram shows flattening of the P and T waves, which may become so small as to be indistinguishable; the QRS complexes generally remain normal; the heart rate is slow; and there may be lengthening of the PR interval up to about 0·24 second, though in many cases the conduction time remains unaffected. T is sometimes inverted (as well as shallow) in lead 1, or in leads 1 and 2, this finding probably implies independent coronary disease (see Figs. 79 and 80)

In rare instances the signs and symptoms of myxoedema heart are found in patients who present neither the appearance nor the other clinical manifestations of myxoedema. On investigation, these patients show a low basal metabolic rate and a raised blood cholesterol The symptoms are relieved and the cardiac enlargement disappears on treatment with thyroid. The condition has been named "Abortive myxoedema" or "Abortive myxoedema heart" by Zondek (1941).

coronary circulation, and this may more than compensate for the extra load thrown on the heart. Most of these patients have an optimum basal metabolism, at which level their cardiac symptoms are minimal; improvement will occur if a metabolic rate originally above the optimum is lowered, or if one below the optimum is raised; and in either case aggravation will occur if the process is carried beyond the optimum level. Independent heart disease, therefore, does not contra-indicate the cautious use of thyroid. Cases should, in the first instance, be treated on the usual lines for the variety of disease present; then thyroid medication may be started. The initial dose should be ¼ grain daily, increased at intervals of a few days while its effects are watched In some cases the symptoms are aggravated from the start, in which case the drug should immediately be discontinued. Many improve on instituting thyroid treatment, and continue to improve until a particular dose is exceeded, when they become aggravated, an effort should be made to ascertain the optimum dose for each particular patient, and the dosage should be maintained at that level In some cases the cardiac symptoms appear to be unaffected by the thyroid, but the general symptoms improve, and in them there is no reason for withholding thyroid The only contra-indication to the cautious trial of thyroid by the method outlined is during the first four to six weeks after a coronary thrombosis

3 The Cardiovascular System in Diabetes and in Hypoglycaemia

Diabetes in young persons does not give rise to changes in the heart or blood vessels The circulatory failure which occurs in ketosis or coma is a peripheral failure due in part to toxic effects on the capillaries, in part to dehydration The circulatory manifestations of hypoglycaemia are associated with peripheral vaso-dilatation, increased circulation rate, and increased cardiac output, the pulse is accelerated and the systolic blood pressure raised, extrasystoles may occur The effects resemble those of an injection of adrenaline. The increase in output sometimes persists for several hours after the hypoglycaemia has been relieved by administration of glucose

should not be given unless congestive symptoms persist despite thyroid therapy.

Myxoedema with Independent Heart Disease.—Many cases of myxoedema are complicated by arteriosclerosis, by coronary disease, or by hypertension. There may be angina of effort, there may be *attacks of coronary occlusion, or* there may be hypertensive heart failure. The symptoms and signs do not

A B

FIG 80—Cardiogram in myxoedema (*contd*).

A *Another case, before treatment*
B *Same patient, after 20 days' treatment with thyroid*

differ from those produced by corresponding lesions in non-myxoedematous patients

In such cases the question of thyroid therapy may be difficult to decide It might be expected that thyroid would aggravate the cardiovascular symptoms by increasing the metabolic rate and throwing more work on the heart, and in fact some cases of coronary disease and some of cardiac failure have been treated by thyroidectomy with a view to lowering the metabolism and the load in the heart While it is true that lowering of the metabolic rate will often produce considerable benefit in non-myxoedematous patients suffering from the condition specified, recent experience has shown that a myxoedematous *patient suffering from a similar complaint* may derive considerable benefit from thyroid therapy. It seems that thyroid, by increasing the circulation rate, improves the

without insulin if it is at all possible to prevent ketosis by dietetic treatment alone.

The second problem which arises is that of fluids when there is coma combined with cardiac failure. Unfortunately this combination is not rare ; for ketosis in itself causes further impairment of cardiac efficiency, and the consequent circulatory failure aggravates the ketosis Now the treatment of diabetic ketosis requires administration of fluid in addition to insulin and glucose, and the fluid is better given intravenously ; but large amounts of fluid, and especially intravenous fluid, will merely add to cardiac oedema and increase the risk of pulmonary oedema. In the circumstances, treatment must be a matter of compromise, the quantity allowed being gauged for each individual case in accordance with the relative severity of the ketosis and the cardiac symptoms ; as might be expected, the result is frequently unsatisfactory.

4 The Cardiovascular System in Disorders of the Pituitary and Suprarenal Glands

Diseases of the pituitary are not infrequently associated with cardiovascular disorders. In *Cushing's syndrome* (pituitary basophilism) hypertension is one of the relatively constant features It may be of a benign type, but occasionally presents the features of a malignant hypertension *Acromegaly* is associated with cardiac enlargement, there may be either hypertension or hypotension. *Simmonds' disease* is usually associated with a slow pulse and low blood pressure Syncopal attacks with hypoglycaemia occur, they resemble the crises of Addison's disease, and may be fatal Some cases show a close resemblance to myxoedema An association has been described between the *Frolich type of hypopituitarism* and congenital cardiac lesions, I have encountered two such cases

The hypotension of *Addison's disease* is well known as one of its most constant features In the *crises* of this disease there is a further drop in blood pressure with all the circulatory manifestations of shock, the superficial veins are collapsed and the blood is viscous as the result of dehydration The crises are attributed to loss of sodium and chloride from the blood with secondary loss of fluid and diminution in the circulating blood volume Improvement has been known to follow

circulation rate accompanying hypoglycaemia does not give rise to anginal pain or to cardiac failure but may be associated with complaint of palpitation.

In marked contrast to the normality of the circulatory organs in young diabetics are the findings in elderly diabetics, a high proportion of whom suffer from hypertension or arteriosclerosis. The incidence of these conditions in diabetics is considerably in excess of their incidence in the general population, large though that is. Angina of effort and coronary thrombosis are frequently encountered, hypertensive heart failure a little less often, and auricular fibrillation with congestive failure occasionally. Hypoglycaemia in an individual with pre-existing cardiovascular disease frequently provokes anginal attacks, and coronary occlusion has been known to follow, less often, hypoglycaemia induces cardiac failure The causative hypoglycaemia is readily overlooked. it should be suspected when angina follows an injection of insulin and should be considered as a possibility when angina or cardiac failure appear in any patient who is on insulin treatment The blood sugar occasionally shows violent fluctuations, particularly in mild diabetics, the fact that a patient's blood sugar is usually at a level of 150 to 200 mg. per 100 ml does not exclude the possibility of hypoglycaemic attacks, even though insulin is not being administered.

The treatment of diabetic cardiac patients is a matter of some importance, and there is more in it than a mere summation of cardiac and diabetic treatment. Each may require modification in special circumstances. Glucose is an excellent cardiac tonic (provided it can be utilised), and hypoglycaemia, while it may cause little damage to a healthy heart, has a deleterious effect on a diseased heart, especially if frequently repeated For these reasons, in treating a cardiac diabetic, it is better to maintain the blood sugar at a rather higher level than would be considered satisfactory in a non-cardiac diabetic, by so doing, the possibility of hypoglycaemia is reduced, and the tonic effect of glucose is obtained provided the patient is able to utilise it. At the same time it is even more important to avoid ketosis than in the non-cardiac diabetic Cardiac diabetics, therefore should be stabilised at a somewhat higher level than their non-cardiac counterparts, and they require more careful supervision. Elderly diabetics are usually better

amounting to 60 mm. If the diagnosis can be established, treatment is surgical. After operation, desoxycorticosterone acetate may be necessary for a time.

5. The Heart in Vitamin B_1 Deficiency

Cardiac failure of congestive type is an important feature in some cases of **beri-beri** ("wet beri-beri"). Pathologically there is dilatation and hypertrophy involving especially the right side, and the heart muscle may show oedema with fatty or other degenerative changes. Clinically, cardiac symptoms may predominate from the onset, alternatively, they may supervene either suddenly or gradually after a period during which the symptoms have been neuritic. The symptoms are those of right-sided (congestive) failure—breathlessness, tachycardia, oedema, enlargement of the liver, oliguria, and albuminuria. Right-sided cardiac enlargement can sometimes be demonstrated clinically, and can more easily be shown radiologically. The first heart sound is often shortened ("tic-tac rhythm" or "embryocardia"), gallop rhythm is frequently present; or a systolic murmur may be heard in the tricuspid area. Electrocardiograms sometimes show little abnormal, but frequently there are changes in the RT and T segments which may become flattened and dome shaped, or may show transient inversion somewhat reminiscent of that seen in pericarditis: the PR interval tends to be shortened, and right axial deviation is frequent, often with inversion of T3. Heart failure in beri-beri occurs with an increased cardiac output.

In **chronic alcoholism** cardiac symptoms are not uncommon. It is now recognised that alcoholism is often associated with defective absorption of vitamin B_1, and the modern tendency is to attribute alcoholic neuritis to this cause ("alcoholic beri-beri") as well as some of the cardiovascular manifestations ("alcoholic beri-beri heart"). In addition, symptoms resembling beri-beri have occurred in cases of dietetic deficiency other than those due to alcoholism ("secondary beri-beri"), for example, in patients suffering from gastro-intestinal disease. Finally, similar findings have been reported in pellagra and have been attributed to simultaneous deficiency of vitamin B_1. In all these circumstances the cardiac features are those of right-sided failure comparable to that occurring in true beri-

administration of sodium chloride even without desoxycorticosterone acetate; preferably, however, the latter drug should be given in doses of 5 mg. every 8 hours in addition to intravenous glucose-saline; the glucose is added, as there is often a hypoglycaemia during the attack.

Of greater interest from the cardiological point of view is the *paroxysmal hypertension* of *suprarenal medullary tumours* (phaeochromocytomata). In this condition there are paroxysmal attacks of hypertension associated with symptoms which are sometimes dramatic The paroxysm is often brought on by change of posture such as to compress the suprarenal tumour, for example by turning on one side, or sometimes by effort, at times they occur without obvious cause The blood pressure in an attack can reach very high levels—usually above 200 and at times reaching 300 mm. Hg. systolic pressure with a corresponding rise in the diastolic pressure. The attack may be associated with severe headache, vomiting, sweating, tremors, muscular twitchings, cramps, and dizziness sometimes there is alarming collapse, which may prove fatal, tachycardia is often a feature of the attack. Minor paroxysms sometimes occur without subjective symptoms, being discovered accidentally on routine blood pressure estimation In the earlier stages blood pressure is normal in the intervals between the paroxysms, but, if unrecognised and untreated, the condition progresses to a permanent hypertension The tumour is often as large as an orange, and may be palpable in one or other loin, or it may so displace the kidney that this becomes palpable in the loin or beneath the costal margin anteriorly. Occasionally the tumours are bilateral Intravenous pyelography shows the downward displacement of the kidney It is stated that the adrenaline content of the blood is much raised, even between the paroxysms, this can best be demonstrated by a biological test, as the chemical estimation of adrenaline presents many difficulties. In a suspected case, seen when the blood pressure is normal, intravenous injection of 0 025 mg histamine in 0 25 ml. normal saline will provoke a typical paroxysm. In persistent hypertension due to phaeochromocytoma injection of piperi-dimethyl-benzo-dioxane ("933 F") in dosage of 10 mg. per square metre of body surface, while giving a slow intravenous glucose-saline drip, produces a fall in blood pressure lasting for 13 to 14 minutes and sometimes

amounting to 60 mm. If the diagnosis can be established, treatment is surgical. After operation, desoxycorticosterone acetate may be necessary for a time

5 The Heart in Vitamin B_1 Deficiency

Cardiac failure of congestive type is an important feature in some cases of beri-beri ("wet beri-beri"). Pathologically there is dilatation and hypertrophy involving especially the right side; and the heart muscle may show oedema with fatty or other degenerative changes. Clinically, cardiac symptoms may predominate from the onset, alternatively, they may supervene either suddenly or gradually after a period during which the symptoms have been neuritic. The symptoms are those of right-sided (congestive) failure—breathlessness, tachycardia, oedema, enlargement of the liver, oliguria, and albuminuria. Right-sided cardiac enlargement can sometimes be demonstrated clinically, and can more easily be shown radiologically. The first heart sound is often shortened ("tic-tac rhythm" or "embryocardia"), gallop rhythm is frequently present, or a systolic murmur may be heard in the tricuspid area. Electrocardiograms sometimes show little abnormal but frequently there are changes in the RT and T segments which may become flattened and dome-shaped, or may show transient inversion somewhat reminiscent of that seen in pericarditis, the PR interval tends to be shortened, and right axial deviation is frequent, often with inversion of T3. Heart failure in beri-beri occurs with an increased cardiac output.

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beri. *It is necessary to point out, however, that chronic* alcoholism is often associated with arterial degeneration or hypertension, and that these may in themselves produce failure (left-sided failure), or they may alter the picture of a pure "beri-beri" failure, producing a mixed type with simultaneous right and left ventricular enlargement

The *diagnosis* should be suggested by the occurrence of *right-sided failure in the absence of the more usual causes of* this condition such as emphysema, mitral stenosis, etc. Gallop rhythm is rare in congestive failure other than that due to beri-beri, and may in itself suggest the diagnosis. The dietary, history, and the occurrence of neuritic signs (e.g. muscle tenderness, loss of tendon jerks) confirm the diagnosis. The final proof is given by the response to vitamin B_1 therapy.

Treatment involves absolute rest, careful nursing, and the administration of vitamin B_1 (aneurine, thiamine). In mild cases it may be given orally (5 mg. daily), but in severe or fulminating cases it should be injected intravenously in doses of from 5 to 50 mg. according to the severity, after a few days the dose may be reduced and intravenous therapy may be replaced by the oral route. The cardiographic abnormalities often become more marked for a few days after thiamine treatment is started, this should not be taken as an indication for discontinuing treatment When there is much venous congestion, venesection is of value. In all cases the dietetic deficiency should be corrected, and in alcoholic cases alcohol must be withdrawn.

Infective Polyneuritis.—Although this condition is not thought to be associated with vitamin B_1 deficiency, it seems appropriate at this point to mention that persistent tachycardia is a very frequent finding The tachycardia is usually out of all proportion to the other manifestations of infection such as fever, leucocytosis, or blood sedimentation rate; and it seems likely that it is due to paralysis of the vagus in consequence of neuritis As a rule there is no evidence of cardiac failure; but the tachycardia may persist into convalescence, rendering the latter slow and giving rise to symptoms of neuro-circulatory asthenia. The available evidence seems to show that vitamin B_1 is without effect in cases of infective polyneuritis, though it is usually given. Treatment is a matter of rest and general nursing. *No specific treatment is either possible or necessary*

for the tachycardia, which subsides slowly as the patient recovers or during convalescence

An analogous tachycardia occurs in other conditions in which the vagus is paralysed, for example in consequence of involvement of the vagus nucleus in bulbar paralysis, etc.; I have seen it also in tetanus.

Other Vitamin Deficiencies.—In *scurvy* there are usually no direct cardiovascular complications, but the heart may be affected secondarily in consequence of an anaemia as described in the following section. In rare instances haemopericardium has occurred as a complication *Rickets* does not give rise to cardiovascular disturbances except in cases where it has produced a rachitic chest deformity (Chapter 16) When cardiac failure occurs in *pellagra*, it is attributed to simultaneous deficiency of vitamin B_1

6. The Cardiovascular System in Anaemias

Cardiovascular symptoms are invariably present in patients who exhibit any significant degree of anaemia. In most cases, however, they are due to the anaemia *per se*, and only rarely do they become severe. There are two reasons why anaemia should cause cardiovascular symptoms. In the first place *a fall* in haemoglobin content of the blood involves a reduction in its oxygen-carrying capacity, this implies that a larger amount of blood is required to yield a given quantity of oxygen. The resting circulation rate and cardiac output are raised. During muscular exercise, a greater increase in circulation rate is necessary in the anaemic patient than in a healthy individual, a moderate effort in the former becomes the equivalent of a severe effort in the latter, and the circulatory reserve is correspondingly reduced Over-exertion on the part of an anaemic patient may be followed by cardiac failure which, in these circumstances, is associated with a raised cardiac output.

In the second place, severe anaemia is associated with malnutrition of the heart muscle Pathologically the muscle is soft while fatty degeneration is found in the individual fibres. In some instances fatty infiltration is also present, an example being the "thrush breast heart" of pernicious

anaemia where the infiltration is visible in the sub-endocardial zone. In other cases of anaemia, pigmentary degeneration ("brown atrophy") has been found. The changes are reversible, and complete recovery is possible. Coronary thrombosis is a rare complication The symptoms vary according to the rapidity with which the anaemia develops and to the presence or absence of independent cardiovascular disease.

Acute Anaemias.—The term "acute anaemia" is here used to describe any form of anaemia which has developed rapidly, examples include acute post-haemorrhagic anaemia resulting from gastro-intestinal or other internal haemorrhage, and the acute haemolytic crises of pernicious anaemia acholuric family jaundice, etc. The onset is marked by sudden pallor and collapse sometimes followed by loss of consciousness; or if consciousness has not been lost, the patients may complain of pain over the heart or of breathlessness The pulse is rapid and feeble, the blood pressure low The heart is of normal size (unless complicating cardiac disease has previously been present), the first sound is short ("tic-tac rhythm"). There is no oedema; on the contrary, signs of dehydration are a feature in the post-haemorrhagic cases, and the circulatory manifestations are those of shock rather than simple anaemia or cardiac failure.

With such a picture it is not surprising that mistaken diagnoses of coronary thrombosis are made from time to time; I have collected six examples in the last year or two in all of which the patient was referred as a case of suspected coronary disease, all being due to gastro-intestinal haemorrhage With sudden collapse at the onset, the possibility of gastro-intestinal haemorrhage should be borne in mind, a history of previous dyspepsia should be enquired into, and the first stool passed by the patient should be inspected for the presence of melaena. Thirst is a suggestive symptom

The *treatment* in these cases is that of shock and dehydration Rest in bed is essential Warmth should be applied, and morphine given to allay restlessness and to facilitate arrest of the haemorrhage. Shock and dehydration are best treated by the administration of fluid this may take the form of an intravenous drip saline or an intravenous drip transfusion. Water may be given by mouth at frequent intervals, this is infinitely more effective in relieving thirst than the old-fashioned

"sips of water and ice to suck", and even in cases of haematemesis the danger of increasing the haemorrhage is almost non-existent.

Chronic Anaemias.—The term "chronic anaemia" is here used to describe any form of anaemia which has developed gradually; it includes pernicious anaemia, the microcytic hypochromic anaemias, aplastic anaemias, and many others.

In the minor grades of anaemia, the usual cardiovascular complaints are of palpitation and breathlessness on exertion; faintness, light-headedness, and dizziness are also frequent. As the severity of the anaemia increases, breathlessness tends to predominate over the remaining symptoms and oedema of the ankles frequently appears. Even in the presence of oedema, enlargement of the liver is uncommon, a point which suggests that the symptoms are due to the anaemia as such rather than to congestive failure from its effects on the heart. Albuminuria, though frequent, is usually slight.

In some cases angina of effort is the outstanding complaint; indeed it may be the only complaint. Alternatively oedema and other congestive symptoms occasionally follow a period during which there has been angina of increasing severity. Less often, the picture in the advanced stage may suggest a left ventricular failure in that Cheyne-Stokes respiration develops along with confusion or delirium; pulmonary congestion, however, is usually conspicuously absent, a point which may be of diagnostic importance. It seems likely that these atypical cases, characterised by anginal pain or symptoms suggesting left ventricular failure, are due to coincident but independent heart disease. This may be insufficient to cause symptoms of failure by itself, yet capable of modifying the picture caused by the anaemia. Recovery follows cure of the anaemia and all symptoms disappear. The possibility that the independent heart disease will slowly progress and that the symptoms will recur after a lapse of years must always be borne in mind. The majority of the patients in this category are over the age of 40, that is, they have reached the "arteriosclerotic age". The following case illustrates this type.

A railway clerk, aged 54, had to cross a hill on his way to work. A year before I saw him he began to have sternal pain radiating to his left arm when ascending this hill, and he would have to stop for a few minutes; gradually the stops became

more frequent so that he began to make a detour round the hill. His doctor reported that his blood pressure at that time was "*over 200 and up to 240*". *A few months later he had* to give up walking and travel by tram. Then he developed oedema of the legs for which he was given Nativelle's Digitaline; this had *no effect on the oedema but he began to have attacks* of anginal pain in the absence of effort; at this stage his blood pressure was said to be "165". I saw him some six months

FIG 81 —Teleradiogram in a case of pernicious anaemia which simulated hypertensive heart failure Male, aged 54 For description of case history see text, p. 381 Note absence of hypertensive aortic or cardiac shadow and absence of pulmonary congestion

later. He was pale and had gross oedema of the legs. He took frequent spasms of sternal pain, during which he would rise from his bed and stand erect, as he said this gave relief He had Cheyne-Stokes respiration, and when admitted to hospital it was noted that he became confused towards evening and was delirious at night. So far the case resembles a typical hypertensive heart failure, but there was no enlargement of his heart and no pulmonary congestion either *clinically* or radiologically (Fig 81); the liver was only just palpable at the height of inspiration The blood pressure was 134/62. The cardiogram (*Fig 47, B, p 173*) *showed low* voltage

QRS deflections and a digitalis effect. Blood examination showed a severe pernicious anaemia (RBC 890,000 Hbg. 25 per cent), fractional test meal and barium meal carried out later revealed complete achlorhydria but no other lesion of the gastro intestinal tract. He made a rapid response to liver therapy, and left hospital free from symptoms six weeks later. Five years afterwards he survived a coronary occlusion, and six months after this he was again seen with hypertension and angina of effort.

Physical signs It is important to note that pallor is unreliable as a sign of anaemia, it occurs in many conditions other than anaemia, and it is slight or absent in some cases with a haemoglobin level as low as 50 per cent. Pallor of the conjunctivae is less unreliable than pallor of the skin or lips, but even here the appearances are sometimes misleading Estimation of the haemoglobin should always be carried out in a pale patient, or in a patient who complains of cardiovascular symptoms for which there is no obvious cause, which are atypical, or which seem disproportionate to the extent of the cardiac damage present. Estimation of haemoglobin is easily and quickly carried out, and it will serve to show whether complete blood examination is necessary

In mild cases of uncomplicated anaemia, the physical findings in the cardiovascular system are often entirely normal. In a proportion of patients systolic murmurs are heard, often at the base of the heart but sometimes at the apex In most cases the murmurs are soft and blowing, while the first sound is heard along with the murmur, but occasionally the murmur is loud and harsh, completely obscuring the first sound.

In more severe cases of anaemia, the first sound becomes soft, quite apart from any murmurs which may be present. There may be evidence of slight enlargement of the heart clinically and radiologically, but more often this is lacking and the heart is normal in size. Considerable enlargement is rare in the absence of complicating heart disease Congestion of the lungs and enlargement of the liver are unusual, and these may be signs of diagnostic significance. The cardiogram in the earlier stages is normal, and it may remain so throughout, or it may show a drop in voltage, either of T or of QRS, as the severity increases.

In exceptional cases there are other murmurs. Reference

has already been made (p. 61) to two cases in which a typical mitral presystolic murmur was heard in patients with a severe microcytic hypochromic anaemia; in neither was there any radiological evidence of mitral stenosis, and in both the murmur disappeared on recovery from the anaemia. I have recently had under observation a boy of 12 who suffers from aplastic anaemia: when first seen his haemoglobin was in the vicinity of 20 per cent; it has on numerous occasions been raised to levels of 80 to 100 per cent by transfusions, but until recently it has always dropped slowly back to the initial level. When this boy's haemoglobin drops below 40 per cent he has a distinct aortic diastolic murmur in addition to a mitral systolic murmur and a third sound at the apex; on raising the haemoglobin level the aortic diastolic murmur disappears at about 40 to 50 per cent, the third sound vanishes between 60 and 70 per cent, while at 80 per cent or more no murmurs can be heard. He has a normal heart shadow radiologically, and a normal cardiogram

Diagnosis. In the absence of blood examination, anaemia is frequently diagnosed when it does not exist, especially in cases of effort syndrome, less often in thyrotoxicosis, hypertension, active endocarditis, or chronic valvular disease. The reverse error is also possible; the murmur of an anaemia may be mistaken for that of an organic valvular lesion, the symptoms of an internal haemorrhage may be attributed to coronary thrombosis, or those of a chronic anaemia to coronary sclerosis or myocarditis It is important to recognise that anaemia sometimes complicates and aggravates organic heart disease, and that treatment of the anaemia leads to improvement in the circulatory symptoms.

Treatment. Treatment is primarily that of the anaemia; blood examination will disclose whether iron, liver, or transfusions are required. Milder cases can be treated while ambulant, but when the haemoglobin level is below 50 per cent, or if symptoms are severe with a higher level, rest in bed is advisable Cardiovascular symptoms due to anaemia respond to correction of the anaemia but to no other form of treatment.

When there are congestive symptoms with venous distension, the advisability of transfusion requires special consideration; as has been pointed out on p. 2, a further rise in the right auricular pressure in these circumstances may

provoke a fall in cardiac output and precipitate acute heart failure, fatal results have been recorded. If it is decided to give a transfusion at all it would appear to be wise to limit the quantity to 250 or 300 c.c., and to give it very slowly.

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CHAPTER 18

TRAUMATIC CARDIOVASCULAR LESIONS

THE heart may be injured by direct or by indirect violence. Traumatic cardiac lesions are rare in comparison, on the one hand with heart disease due to natural causes, and on the other with accidents as a whole, yet some knowledge of their effects is important to enable the practitioner to avoid alike the errors of overlooking genuine cardiac injury, or of creating a cardiac neurosis. A comprehensive review of the subject of cardiac trauma has been made by Barber (1944), from which I have received much assistance in the compilation of this chapter.

For descriptive purposes traumatic heart lesions may be considered under the following headings:

(1) Penetrating wounds.
(2) Bruising (contusion) of the myocardium or pericardium from blows on the chest or from crushing injuries.
(3) Valvular lesions resulting from direct or indirect violence.
(4) Coronary lesions resulting from trauma.
(5) "Primary cardiac strain."
(6) Arrhythmias following trauma
(7) Effects of trauma in persons with pre-existing heart disease.
(8) Cardiac neurosis following trauma
(9) Arteriovenous aneurysm.

1. PENETRATING WOUNDS OF THE HEART

Penetrating wounds are commonly the result of stab injuries or bullet wounds: less often they arise in consequence of fracture of ribs. When there is free communication either with the pleural cavity or with the outside, death usually supervenes quickly. When the pericardial wound closes, death may result from accumulation of blood in the pericardium (haemopericardium); but recently certain recoveries have been reported.

The chief symptoms are those of cardiac tamponade with a fall in blood pressure and pulse pressure. The skin is cold and

dusky, often sweating. The veins in the neck are distended. There is a tendency to bradycardia. The cardiac dullness is increased, while the cardiac impulse and heart sounds are feeble. X-ray examination shows enlargement of the heart shadow, the shape being that of a pericardial effusion, and the pulsations are feeble. The cardiogram resembles that of a pericardial effusion

Recoveries have been reported in cases where the wound was sutured Bullock (1936) quotes a case in which a wound in the right ventricle was successfully sutured although the patient's heart had ceased to beat before the operation reached the pericardium, nine years later the patient was in good health. It is suggested that suture should be attempted even though the heart has ceased to beat for a few minutes, or perhaps up to a period of half an hour More recently, Blalock and Ravitch have recommended aspiration of the effused blood in preference to suture, operation being performed only if the bleeding recurs.

Foreign bodies, such as bullets or fragments of shrapnel in the myocardium, may become encysted, causing little inconvenience In such circumstances the foreign body should be left alone On the other hand a foreign body in the myocardium may cause symptoms similar to those of a myocardial contusion or of cardiac infarction, viz. attacks of pain, palpitation, or dizziness Cardiographic changes may or may not be present in occasional cases abnormalities in rhythm have been reported When a foreign body produces symptoms, excision should be considered

2 Bruising (Contusion) of the Myocardium or Pericardium

Aetiology and pathology Contusion of the myocardium or pericardium is most often the result of a crushing injury, but it may also follow a blow over the heart Fracture of ribs or sternum is not necessarily present, and even bruising of the superficial tissues may be absent A common example is the "steering-column accident" in which a driver's chest comes in violent contact with the steering column during a collision. Cardiac damage has also followed less severe blows, for example, a blow over the heart with a cricket ball, Moullin

instances a case in which a haemopericardium resulted from a knock over the sternum from a player's elbow during a game of football

The myocardial lesions may involve any of the four chambers and may be single or multiple. A blow on the front of the chest sometimes produces a lesion on the posterior surface of the heart. A frequent site is on the posterior surface of the right auricle near the entrance of the inferior vena cava in the pericardio-phrenic angle, such a lesion possibly explains the occasional development of abnormalities of rhythm after accidents of the type in question.

The bruising may extend from the pericardial surface inwards, or from the endocardial surface outwards. The latter type is liable to rupture after a delay of some days Lesions of the left ventricle seem less likely to be fatal than those of the remaining chambers Bruising of the pericardium may give rise to a simple (sterile) pericarditis with transient friction; more often there is haemorrhage into the pericardium (haemopericardium) with symptoms and signs similar to those resulting from stab wounds. Secondary pyogenic infection has been described and has been known to lead to a fatal purulent pericarditis. Pneumopericardium is a rare sequel.

Experimental evidence in dogs suggests that when recovery occurs, the bruised area is converted into a fibrous scar indistinguishable from that which results from a myocardial infarction Pericardial adhesions are usually present. In a number of human cases symptoms suggestive of myocardial contusion have been followed by recovery A late sequel in occasional cases has been the development of a cardiac aneurysm. Rupture of the interventricular septum has also been described as a sequel to contusion (East, 1946)

Symptoms and signs Myocardial bruising may be a "silent" lesion; Barber suggests that symptomless cases are by no means rare. Frequently the patient's symptoms are due to concomitant lesions elsewhere, and in such circumstances cardiac damage is recognised by the discovery of pericardial friction or of cardiographic changes on routine examination. At the opposite extreme, severe bruising may be immediately fatal, either from ventricular fibrillation or from rupture of the heart.

In cases with bruising of intermediate severity, cardiac symptoms arise. A striking feature in many cases has been

the presence of a latent period between the accident and the onset of symptoms; the interval varies from 20 minutes up to 3 days, but is often about 12 hours. After this latent interval, the development of symptoms is often rapid, or even abrupt. The symptoms take the form either of severe breathlessness with orthopnoea, of acute pulmonary oedema, or of anginal pain with signs of shock, faintness and low blood pressure. On physical examination there may be pericardial friction, there may be signs of a haemopericardium (p. 193), or the splashing sound of a pneumopericardium may be recognised; more often, however, there are simply short, faint heart sounds. Where the interventricular septum has ruptured a murmur identical with that occurring in congenital ventricular septal defect has appeared. Cardiographic changes are usually present, though they vary in type; most convincing are those in which the changes are similar to those of coronary occlusion; sometimes the cardiogram is that of pericarditis; other findings reported have been flattening, inversion, or exaggeration of the T waves, enlargement of the Q waves, and slurring or notching of the QRS complexes.

In favourable cases improvement sets in quickly, but relapse may occur if the period of rest is insufficient. In a number of cases the patient has become able for all ordinary exercise. In some, angina of effort has appeared during convalescence, while in others shortness of breath and dizziness have remained as permanent sequelae. The differentiation of the latter from cases of neurosis (effort syndrome) may be difficult, especially if cardiographic and radiological evidence of myocardial damage disappears during convalescence. Cases are on record, however, in which breathlessness and dizziness remained, though full investigation of the cardiovascular system revealed no abnormality while the patient did not show the variability, the sweating, or the nervousness of the usual psychoneurotic case. A rare sequel is the later development of a cardiac aneurysm.

Treatment.—Rest in bed is undoubtedly the most important measure in the treatment of a myocardial contusion. [illegible] suggest [illegible]

[illegible] the effused blood should be aspirated. Convalescence should be slow and carefully graduated.

3. Traumatic Valvular Lesions

Valvular rupture may result from direct or from indirect violence. While healthy valves may be ruptured, those which are the seat of pre-existing disease are more likely to be affected.

Rupture of the aortic valve is the most frequent traumatic valvular lesion. It may result from a blow on the chest or from the strain of some exceptional effort ; a diseased aortic valve is liable to rupture in response to a less severe strain. The onset of signs and symptoms is almost invariably immediate , but I have recently seen a patient in whom the symptoms and signs of a ruptured aortic valve appeared 24 hours after a "steering-column accident" : presumably in this case the primary lesion was a contusion of the interventricular septum close to the site of attachment of the aortic valve cusps, the secondary rupture of which must be attributed to extension of the contused area. In the more usual case there is immediate distress with breathlessness , pain, dizziness, pallor, or collapse are other frequent symptoms A loud aortic diastolic murmur appears, sometimes both systolic and diastolic murmurs. There is an immediate fall in the diastolic blood pressure. The accident is followed by dilatation of the left ventricle which may be progressive, and by development of the remaining signs of gross aortic regurgitation (p. 216). Heart failure follows, and usually runs a downward course towards a fatal termination.

Rupture of the mitral valve has resulted from a blow, but a more frequent injury is *rupture of chordae tendineae* This may also follow strain, especially if there has been pre-existing disease of the valve The symptoms consist of immediate pain, breathlessness, signs of shock, and sometimes pulmonary oedema. A mitral systolic thrill and murmur appear, the murmur being loud and conducted to the axilla X-ray examination shows an enlarged pulsating left auricle Cardiographic findings are not characteristic. Heart failure and auricular fibrillation usually follow, though the downhill progress may be slow. An instance with recovery has been reported by Anderson (1940) The *papillary muscle of the mitral valve* may rupture in similar circumstances or following a contusion, in which case the onset of symptoms may be delayed. The signs and symptoms are the same as those of rupture of the chordae tendineae

Rupture of the interventricular septum after a "steering-column accident" has been recorded by East (1946). In this case the sequence of events was probably a contusion followed by later rupture. No murmur was noted in hospital during the two weeks immediately after the injury: but a thrill and a loud murmur with the characteristics of the "bruit de Roger" appeared on the patient's return home, the signs were unchanged three and a half years later.

In the vast majority of cases traumatic valve lesions result in regurgitation at the affected valve, and they tend to run a downward course to a fatal termination. White and Glendy (1941) state that "stenosis has never yet been shown to be of traumatic origin". This statement probably requires qualification in the light of a case quoted by Barber, the development of stenosis in a traumatically injured mitral valve was observed over a period of ten years, and was ultimately confirmed at autopsy twenty-two years after the original injury.

4 Coronary Lesions Resulting from Trauma

It is by no means uncommon for an attack of coronary thrombosis or for onset of effort angina to follow an accident. A number of authentic cases have been reported in which coronary thrombosis has followed direct violence such as might have produced a myocardial contusion. While the mechanism whereby the injury has caused the occlusion may be obscure, and the differentiation between the symptoms of contusion and those of infarction may be difficult or impossible, few would be prepared to deny a causal relationship between the accident and the attack It is quite conceivable that bruising in the neighbourhood of a coronary twig might precipitate thrombosis, especially if atheroma were present. Most of the persons affected have been elderly and may be presumed to have had pre-existing atheroma

When the accident is such that there has been no direct violence, but indirect violence or "strain" is alleged as the cause of the attack, a decision is more difficult This question as it relates to individual cases has been considered in the section on coronary thrombosis (p. 321), and is further discussed in section 7 of the present chapter

When effort angina develops after an accident, similar

considerations apply. I have already pointed out that abrupt onset of effort angina should be diagnosed and treated as a mild attack of coronary occlusion (pp. 323 and 341)

5. Primary Cardiac Strain

The subject of "primary cardiac strain" is one on which there is still divergence of opinion among cardiologists. All are agreed that excessive muscular effort is capable of damaging a diseased heart and of causing deterioration of the cardiac reserve, a breakdown in compensation, or an arrhythmia; this, however, is not primary cardiac strain, and it is discussed in connection with the effect of trauma in persons with pre-existing heart disease (section 7). Opinion is sharply divided on the question as to whether strain or effort can damage a healthy myocardium, which is the implication of the term "primary cardiac strain" Most cardiologists agree that a healthy heart will certainly not be affected by any ordinary strain such, for example, as is involved by strenuous games or by heavy work. The question only arises in cases of quite exceptional strain

Bramwell (1931) found cardiac hypertrophy, often with bradycardia, in Olympic marathon runners, but no corresponding hypertrophy in other Olympic competitors, for example, sprinters The hypertrophy in the marathon runners is not accompanied by any evidence of disease, nor by any reduction in the efficiency of the heart, on the contrary, the reverse is the case: whether such hypertrophy affects the individual's expectation of life remains to be seen. These cases, in which the findings appear to represent a physiological hypertrophy in response to prolonged and frequently repeated muscular strain, and in which the heart is functionally efficient, clearly do not merit the designation of "primary cardiac strain" which implies that damage has resulted and that functional efficiency has been impaired

While it is true that the literature contains occasional examples in which the history suggests that a healthy heart has been rendered inefficient in consequence of severe muscular effort, there is always room for an element of doubt as to the previous state of the heart. Personally I am among those who believe that a perfectly healthy heart cannot be damaged by

muscular effort or strain (apart from rupture of valve cusps described above). I believe that Nature has made provision whereby the voluntary muscles become fatigued before effort reaches the degree at which it endangers the myocardium; and that whenever cardiac damage appears to have followed strain there has been some other pre-existing factor which has lowered the patient's cardiac reserve.

The practical implication of this view is that primary cardiac strain should not be lightly diagnosed. Before doing so the patient's general health should be subjected to a searching examination, especial care being taken to seek sepsis, anaemia, and psychoneurosis as well as to exclude organic cardiovascular disease Failure to discover some such condition which is probably the true explanation for the symptoms, leads to the erroneous diagnosis that a healthy heart has been "strained" during the course of some normal effort such, for example, as playing rugby. This diagnosis is very likely to lead to effort syndrome or to a psychoneurosis which may mar the patient's health for many years to come I have seen a great many cases in which the erroneous diagnosis of a "strained heart" during childhood or adolescence has led to years of unnecessary invalidism

6 Arrhythmias Resulting from Trauma

While any arrhythmia may follow trauma or strain, **auricular fibrillation** is most frequently encountered Though more likely to follow strain in a diseased than in a healthy heart, several cases have occurred following exceptional effort in persons with apparently healthy hearts. Acute discomfort and breathlessness develop during or just after, the effort, and normal rhythm can be restored by quinidine. When fibrillation follows direct violence, the possibility of contusion of the right auricle arises, in some such cases a tendency to congestive failure has persisted The use of quinidine in a case of myocardial contusion might well be attended with danger (cf coronary thrombosis and see p 139), where there has been direct violence, a period of rest and observation is indicated, and it is wiser to withhold quinidine for a few weeks In many accidents there is considerable excitement, and this provides an additional mechanism whereby auricular fibrillation might

be induced. In some accidents all three factors (strain, excitement, and bruising) are present, and it may be impossible to determine which is responsible for the arrhythmia.

Auricular Flutter *is considerably less frequent than auricular* fibrillation, but is of similar aetiology. Identical considerations apply in the matter of treatment.

Paroxysmal tachycardia followed direct trauma in two cases in Barber's series, while ventricular tachycardia has been reported by White and Glendy. The latter authors suggest quinidine, but the wisdom of this might be questioned on the same grounds as in auricular fibrillation

Heart block is a less frequent sequel of trauma In about a third of the published cases it has been transient, in the remainder permanent and occasionally fatal . Usually there has been direct violence and a contusion involving the bundle may be assumed , but in one case complete heart block was recorded in a man with a piece of shrapnel embedded near the apex of the heart and nowhere near the Bundle of His (Lea, 1917). Heart block following indirect violence seems to have been described on one occasion only (Tuohy and Boman), and is not easy to explain.

Extrasystoles occur in a number of cases after accidents. As has been pointed out in the discussion on the aetiology of extrasystoles (p 109), these are often due to extracardiac causes, excitement or other nervous disorder is probably responsible for the majority of cases in which extrasystoles follow an accident On the other hand, multi-focal extrasystoles, extrasystoles occurring in runs, extrasystoles which persist with a rapid heart rate, or extrasystoles which become more frequent after exercise might reasonably be regarded as evidence of myocardial damage

Ventricular fibrillation has been shown to be the cause of death in animals subjected to experimental trauma, and may be assumed when immediate death results from a blow over the heart and when post-mortem examination reveals no other explanation of the sudden death

7. Effects of Trauma in Persons with Pre-existing Heart Disease

It has been pointed out in the preceding sections that certain forms of cardiac damage are more *likely to occur in*

persons who are the subjects of pre-existing heart disease, examples include valvular rupture in those with diseased valves, auricular fibrillation in those with mitral stenosis or with chronic myocardial disease, and angina of effort or coronary occlusion in those with pre-existing atheroma When an accident has involved direct violence it is usually not difficult to decide from the clinical history whether additional damage to the heart has resulted. Valvular rupture and auricular fibrillation are associated with immediate exacerbation of symptoms, with appearance of additional physical signs, and with abrupt diminution of cardiac reserve or development of heart failure In the case of a myocardial contusion, the onset of symptoms may be delayed for a few hours, but when they do appear the onset is abrupt and the manifestations are dramatic

The decision is infinitely more difficult when a patient who has been partially incapacitated by heart disease alleges that his condition has been aggravated by a particular strain, for example, in lifting a heavy weight or from some minor accident In these circumstances certain guiding principles may be of assistance, these involve enquiry as to the severity of the strain, whether this was a routine effort or an exceptional effort for that particular patient and whether the onset of symptoms was immediate or delayed.

Firstly, muscular effort which is beyond the capacity of a patient's heart may initiate cardiac failure or provoke an abnormal rhythm Therefore *if the strain in question has been exceptional for that particular patient* and *if it has resulted in immediate symptoms* it is reasonable to attribute the onset of failure to the strain

Secondly, there is no good evidence that muscular effort can exert a delayed effect in producing cardiac failure Any such suggestion is contrary to the known facts regarding the behaviour of the heart and circulation during rest and exercise It follows that *a strain whether exceptional or routine, which has not produced immediate symptoms*, cannot be held accountable for symptoms arising after a lapse of hours, days, or weeks.

Thirdly patients with stationary cardiac lesions do not show violent fluctuation in their capacity for effort from day to day in the absence of complications There may be gradual improvement in a patient who is convalescent from acute heart

disease, or gradual deterioration in one whose heart disease is progressive. In the latter circumstances the onset of symptoms is gradual; a particular effort first causes no breathlessness, then slight breathlessness, gradually becoming more severe over a period of weeks or months until finally the patient is totally unable to carry out that particular effort. Sudden deterioration in the cardiac reserve indicates that some complication has arisen; it may be an extension of the cardiac disease (e.g. involvement of an additional branch of a coronary vessel); it may be an incidental infection (e g. a cold or a sore throat); it may be an abnormal rhythm, or a lung infarct, or an anaemia, or a dyspepsia, or an anxiety state (see p. 10). In such circumstances an effort which was undertaken one day without any symptoms produces more or less severe breathlessness next day; the patient is apt to blame the effort for "aggravating" his heart disease, whereas the true state of affairs is that the deterioration was already present when the effort was undertaken. It follows, therefore that cardiac symptoms developing during *an effort which is a routine effort for that particular patient* and which has failed to produce symptoms on previous occasions are not attributable to the effort. In these circumstances it can be stated positively that the deterioration preceded the effort in question

It must be borne in mind that patients with organic heart disease are not immune to anxiety. An accident often arouses fear that the heart condition has been aggravated, and so leads to an anxiety neurosis as a complication of the original heart disease

8 Cardiac Neurosis Following Trauma

Neurosis is undoubtedly the most frequent sequel to accidents encountered in cardiological practice. It arises especially in cases which are the subject of compensation or litigation. It is usually traceable to fear of cardiac damage or to anxiety regarding future earning capacity. This may be engendered by the nature of the original accident by knowledge of other cases in which cardiac symptoms have followed accident, by indiscreet remarks on the part of a medical attendant regarding tachycardia, murmurs, or other physical signs, or even by the wording of an incapacity certificate. Once aroused, it is increased by the procedure under the Workmen's Compensation

Act whereby the patient is examined by several doctors who are either "for him" or "against him"; the former are apt to put the most serious possible complexion on his symptoms, and the patient naturally believes them; while he instinctively mistrusts those "against him" who try to minimise or explain away his symptoms.

The actual symptoms may take the form of a neurocirculatory asthenia with tachycardia, of an effort syndrome, or of attacks of nervous precordial pain; they are apt to vary in severity from day to day; they are accompanied by insomnia, nervousness, tremors, sweatings, and other signs of nervous instability. They do not differ from the symptoms of neurosis as found in non-accident cases, described in detail in Chapter 20 (p 433).

While it is important not to overlook genuine cardiac trauma, it is equally important to avoid creating a neurosis and to take steps for its prevention from the start of treatment in an accident case. The appropriate treatment is discussed in Chapter 20. In the special circumstances which exist in compensation cases, the responsibility for prevention and treatment must rest on the patient's family doctor, or on a specialist called in by the latter for that purpose. A practitioner called in by the employers or by an insurance company is unlikely to gain the patient's confidence sufficiently to be able to exert any therapeutic influence. It is also important that the question of liability and compensation should be settled finally at the earliest possible moment.

9. Arteriovenous Fistula or Aneurysm

Arteriovenous fistulae are usually traumatic in origin, though occasionally they arise in consequence of disease processes. Whatever their origin, they have an important effect on the circulation and they are capable of causing heart failure which can be relieved by surgical closure of the fistula. The effect of the fistula is twofold, on the one hand, escape of blood from the artery leads to a fall in the diastolic blood pressure and an effect on the circulation similar to that of aortic regurgitation; on the other hand, entry of blood into the vein at high pressure greatly increases the venous return to the right side of the heart and increases the work of the whole heart.

Cardiac failure develops sooner or later in a high proportion of cases, but the interval before its appearance varies widely, being sometimes less than two years, sometimes as long as nineteen years (Osler, 1915) As a rule the symptoms are those of combined right and left heart failure—breathlessness, pulmonary congestion distended veins, enlarged liver, and oedema; but either right- or left-sided symptoms may predominate. Anginal pain is present in some cases, no doubt due to the low diastolic pressure, and it has been observed to disappear after closure of the fistula (Perthes, 1924). The failure tends to be progressive, and though it may be slow, extending over a number of years it ultimately proves fatal unless the fistula is closed Dramatic recovery follows closure

Physical signs fall into two groups, local and general The local signs are a pulsating swelling over which a purring thrill is palpable and a "machinery" or "humming top" murmur can be heard the murmur is similar to that of a patent ductus arteriosus, being continuous but waxing and waning, loudest in systole Distal to the swelling the arterial pulse is diminished or abolished and there may be venous distension with oedema The general signs are enlargement of the heart involving all chambers, water-hammer pulse with low diastolic blood pressure and moderate tachycardia, capillary pulsation is sometimes seen and the systolic blood pressure may be higher in the legs than in the arms A diagnostic feature is that the pulse slows and the diastolic blood pressure may rise if the aneurysm is compressed

The treatment consists in surgical closure Relief is often dramatic, subjective symptoms improve, the heart diminishes in size, signs of failure clear up the pulse-rate drops, and the blood-pressure figures become more normal Short of closure, compression of the aneurysm gives some relief

10 The Circulation in Paget's Disease

Although Paget's disease of bone is not traumatic in origin, the circulatory manifestations are apparently related to those of arteriovenous aneurysm, and it seems appropriate to consider them at this point. Wide anastomotic channels develop between arterioles and veins in the bones in this disease, and their cumulative effect is similar to that of an arterio-venous

aneurysm. Edholm, Howarth, and McMichael, using plethysmography, have shown that the blood-flow through a humerus affected by Paget's disease may be twenty times greater than that through a normal bone. The flow can be reduced by applying a tourniquet above the point of entry of the nutrient vessel, but not by a tourniquet applied below this level, thereby demonstrating that the increased flow occurs through the bone itself. The work of both ventricles is increased as in the case of arterio-venous aneurysm, and the pulse pressure is raised. Arterial degeneration is found in most cases of Paget's disease who have passed the age of 50, hypertension and retinal arterio-sclerosis sometimes with haemorrhages are present in many. Whether this is a direct consequence of the increased blood flow or merely a co-incidental finding related to the age group must remain a matter of conjecture. Despite the profound effects on the circulation many patients with Paget's disease live for a prolonged period and death is not invariably due to cardiac failure, some dying from intercurrent infection, some from compression paraplegia, and a few from sarcoma of bone. Cardiac failure is nevertheless the most frequent cause of death; it may be either right-sided or left-sided, and it is associated with a greatly increased cardiac output.

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(See *Brit. Heart Jour.*, **7**, p. 212, 1945; also *Clin. Sci.*, **5**, 1944.)

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intensely cyanosed than the face or hands. Conversely, in cases of transposition of the great vessels, an arterio-venous shunt through a patent ductus directs oxygenated blood to the lower extremities, which are therefore less cyanosed than the face or hands.

Approximately 5 gm. of reduced haemoglobin must be present per 100 ml. of blood in order to produce visible cyanosis. Normal blood with 100 per cent haemoglobin content contains 15 gm. of haemoglobin per 100 ml. Thus one third of the haemoglobin must be reduced before cyanosis can be seen. If there is anaemia with a haemoglobin content of 10 gm. per 100 ml., one-half of the haemoglobin must be reduced to give visible cyanosis; whereas with a polycythaemia and 20 gm. haemoglobin per 100 ml. cyanosis can be seen when only one-quarter of the haemoglobin is reduced. Thus anaemia tends to mask and polycythaemia to accentuate cyanosis resulting from a veno-arterial shunt. In an anaemic patient, it may be necessary to estimate the percentage unsaturation of arterial blood in order to detect a veno-arterial shunt.

The cyanosis of congenital heart disease is usually followed by changes in the ends of the fingers and toes; these become enlarged and bulbous with longitudinal as well as transverse curving of the nails, a condition called *clubbing*. Clubbing occurs in chronic lung diseases (especially bronchiectasis and bronchial carcinoma) and in subacute bacterial endocarditis as well as in congenital heart disease. In some cases there is periosteal thickening of the distal ends of the radius, ulna, tibia, and fibula termed "pulmonary osteo-arthropathy".

Murmurs depend on the passage of blood through a relatively small aperture, or on eddies set up when blood emerges from a small aperture into a larger cavity. A certain rate of blood flow in relation to the size of the aperture is required to produce a murmur. The flow of blood through an aperture depends on the existence of a pressure difference between the two sides of the aperture. With a sufficiently large aperture there will be no murmur; the extreme example of this is *cor triloculare*. With a smaller aperture, a murmur will be heard when there is a sufficient pressure diff

... With a patent ductus

CHAPTER 19

CONGENITAL CARDIAC LESIONS

GENERAL CONSIDERATIONS

Cyanosis in congenital heart disease depends on the existence of a right-to-left shunt ("veno-arterial shunt") whereby venous blood enters the arterial blood stream. Some lesions, notably those in which there is obstruction of the pulmonary valve or pulmonary artery accompanied by an auricular or ventricular septal defect, involve a permanent veno-arterial shunt; the victims are cyanosed from birth. These comprise the *cyanotic group* of congenital malformations. Many congenital lesions involve no shunt, for example, coarctation of the aorta, aortic stenosis, bicuspid aortic valve, and congenital heart block. In these cases there is no cyanosis, and they are known as the *acyanotic group*. Any cyanosis which does appear in the stage of failure is comparable to that of acquired heart disease being attributable to pulmonary congestion, to slowing of the blood stream in the peripheral capillaries, or to a combination of these factors; it is usually much less intense than the cyanosis due to a veno-arterial shunt. A third group of cases is described as *potentially cyanotic*; it includes atrial septal defect, ventricular septal defect, and patent ductus arteriosus. These lesions ordinarily involve an arterio-venous (left-to-right) shunt; arterial blood enters the venous circulation, but there is no flow in the reverse direction and no cyanosis. Ultimately, owing to the development of failure or of some pulmonary complication, the pressure on the right side (auricle, ventricle, or pulmonary artery) rises and exceeds that on the left side (auricle, ventricle, or aorta); the shunt becomes from right to left, and intense cyanosis (the so-called "cyanose tardive") develops.

The distribution of cyanosis in congenital heart disease is sometimes of diagnostic significance. A veno-arterial shunt through a septal defect directs venous blood to all parts of the body, whereas a similar shunt through a patent ductus arteriosus directs the greater part of the venous blood to the descending aorta, so that the lower extremities are more

intensely cyanosed than the face or hands. Conversely, in cases of transposition of the great vessels, an arterio-venous shunt through a patent ductus directs oxygenated blood to the lower extremities, which are therefore less cyanosed than the face or hands

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... With a patent ductus

arteriosus a pressure difference often persists into diastole so that the murmur is continued through systole into diastole, being louder in systole and fainter in diastole (the "machinery murmur", "humming-top murmur" or "Gibson's murmur") With aortic or pulmonary stenosis, certain degrees of narrowing will give rise to a murmur; but if there is complete atresia, there is no murmur

Inasmuch as murmurs depend on a pressure difference as well as on the existence of an aperture of appropriate size, they may vary from time to time in a given patient, they may even disappear should the pressure on the two sides of the aperture become equal. Evidence that a murmur was not present on some previous occasion does not therefore exclude a congenital lesion, nor can disappearance of a murmur be accepted as disproving the existence of a congenital lesion, or as proving the closure of an abnormal communicating channel

Pressure differences between the right and left sides are less in infancy than in childhood or adult life. Furthermore, the small size of an infant's chest renders exact localisation of murmurs less reliable. For these reasons murmurs are of less help in the diagnosis of congenital malformations in infants than in older children or adults.

Combined Congenital and Acquired Heart Disease.—The presence of a congenital lesion does not preclude the development of acquired heart disease Rheumatic mitral stenosis not infrequently appears in persons with an atrial septal defect, this combination being known as *Lutembacher's syndrome*. One of my patients had mitral stenosis complicating a patent ductus arteriosus. Subacute bacterial endocarditis may become engrafted on a congenital lesion (atrial septal defect, ventricular septal defect, patent ductus arteriosus or bicuspid aortic valve); it is one of the chief dangers of these lesions though there is considerable difference of opinion as to its frequency When it complicates a septal defect or patent ductus in the absence of heart failure, the shunt is usually from left to right so that emboli are swept into the venous side of the circulation giving rise to infarcts in the lungs the symptoms consist of recurring lung infarcts with persistent or intermittent fever When bacterial endocarditis complicates these lesions in the stage of failure with a right-to-left shunt, or when it affects a bicuspid aortic valve, the emboli lodge in the systemic circulation

Diagnosis in Congenital Heart Disease.—The history, together with careful physical examination, X-ray, and fluoroscopy, will in many cases reveal the nature of the malformation. Of especial importance is the recognition of the particular cardiac chambers which are enlarged. Dr. Helen Taussig, whose experience of congenital heart is probably unsurpassed by that of any other living physician, places her chief reliance on these methods and confesses that she has little personal experience of angiocardiography. In selected cases the latter adds to the information given by radiology. Cardiograms help chiefly by detecting hypertrophy of one or other ventricle, and also in cases of congenital heart block. Arterio venous shunts can be confirmed by cardiac catheterisation coupled with blood gas analysis (p. 29). Veno-arterial shunts can sometimes be detected by measurements of the circulation time, by passage of the cardiac catheter through a septal defect, or by early filling of the left side of the heart on angiocardiography.

Failure in Congenital Heart Disease.—Failure in congenital heart disease may be right-sided, left-sided, or combined. Pulmonary stenosis and lesions of the pulmonary artery lead to right ventricular hypertrophy and ultimate failure. Aortic stenosis or coarctation of the aorta cause hypertrophy and ultimate failure of the left ventricle. Matters are more complicated with lesions involving shunts. Atrial septal defect leads in the first instance to dilatation of the right auricle: the right ventricle next receives extra blood and becomes overfilled; the pulmonary arteries in turn become dilated, and the venous return to the left auricle itself is increased. Failure is primarily right ventricular: it leads to a rise in the right auricular pressure with reversal of the shunt and intense cyanosis. Ventricular septal defect throws additional work on both ventricles if the amount of blood passing through the aperture is significant; failure may involve both ventricles equally, or either predominantly. With a patent ductus arteriosus the pulmonary artery is overfilled and the pulmonary pressure rises; the venous return to the left auricle and left ventricle is increased, and these in turn become dilated and hypertrophied. At the same time the raised pressure in the pulmonary artery may lead to hypertrophy of the right ventricle. Failure is usually left ventricular but may be combined.

1. Defects of the Interauricular Septum

Patent Foramen Ovale.—The foramen ovale normally closes at birth. Not infrequently the closure is incomplete, leaving a valvular or slit-like opening between the right and left auricles. This is undoubtedly the most frequent of all congenital cardiac anomalies. These minor communications between right and left sides cause no impairment of cardiac efficiency, nor do they give rise to any physical signs which can be recognised during life. They are found not infrequently post-mortem in persons who have never suffered from any cardiovascular symptoms, and they are of no clinical importance.

Atrial Septal Defect.—This condition is developmentally different from simple patency of the foramen ovale; it is associated with a considerably larger communication between the two sides of the heart; it eventually produces cardiac failure, and it can be recognised during life. It is due to failure of development of the septum secundum. A wide aperture in the interauricular septum results; its diameter may be an inch or more. It may occur as an isolated lesion, or in combination with other lesions either congenital or acquired; ventricular septal defect, pulmonary stenosis or mitral stenosis are frequent associated lesions. As long as the pressure in left and right auricles remains equal there is no shunt, and the lesion remains "silent." If the left auricular pressure rises, as it tends to do in adult life, a left to right shunt develops; the right auricle, and next the right ventricle, become overfilled; then the pulmonary arteries are similarly affected. Thus are produced the murmur and the characteristic radiological features. With the onset of failure the right auricular pressure rises while left auricular pressure drops; hence the shunt becomes from right to left and intense cyanosis develops (the "cyanose tardive" of French writers).

Clinically, the symptoms are relatively late in developing, often appearing towards the end of the third or fourth decade. One of my cases, an army major, first developed symptoms when sent on a gas-training course at the age of 54; after a year's sick leave he returned to light duty for another year before being invalided from the army; at the age of 60 he was in the Home Guard, but was finding the duties too arduous

Cyanosis and breathlessness are the main clinical symptoms. There may be no murmur, or there may be a murmur similar to that of ventricular septal defect, that is to say a loud blowing

FIG. 82.—Atrial septal defect

Uncomplicated case. Male aged 60. History and symptoms described in text, p. 404. Note enlarged pulmonary conus, gross dilatation of the descending branch of the right pulmonary artery and of three branches of the left pulmonary artery radiating out from the hilar shadow. The left ventricle is a little enlarged.

systolic murmur, heard over a wide area, but maximal at the sternal end of the fourth left costal cartilage or a little above; occasionally the murmur is presystolic in time.

Cardiograms show partial or complete R. bundle-branch-block. The radiological appearances are diagnostic. There is

well-marked enlargement of the conus pulmonalis and of the larger branches of the pulmonary arteries. On the right side, a large comma-shaped shadow passing downwards from the

Fig. 83 – Atrial septal defect (*contd.*)

A *Atrial septal defect with dextrocardia* (Dr A Glen's case) Antero-posterior view Compare Fig 82, and see Figs 83 B and C

hilum is produced by the distended inferior branch of the right pulmonary artery, with finger-like processes representing the upper and middle branches, on the left side the branches radiate from the hilum and are considerably wider than normal. A striking feature is the absence of congestion of the peripheral parts of the lung fields. On screening, " hilar dance " is seen,

due to pulsation of the distended pulmonary artery branches. The differential diagnosis is from emphysema, in which distension of the pulmonary artery branches is considerably less and hilar dance is also less marked, the clinical signs of

B

Fig. 83.—Atrial septal defect (contd.)

B. Same case as 83 A, right oblique view. This view shows the right-sided aortic arch and prominence of the posterior contour which in this case is formed by the dextroposed left ventricle.

emphysema are absent. In mitral stenosis, congestion involves the periphery of the lung fields first, and to a greater extent than the hila. Primary pulmonary hypertension may give a similar radiological picture. Theoretically a similar picture could result from disease of the pulmonary artery.

Treatment is symptomatic only.

2. Defect of the Interventricular Septum

This is another common congenital anomaly which may occur alone, or in combination with other congenital defects; it is frequent in cases of congenital pulmonary stenosis, and

C

Fig 83—Atrial septal defect (*contd*)

C *Same case as 83, A*, left oblique view The prominent conus pulmonalis is seen anteriorly, while dilated pulmonary vessels radiate backwards

together with this lesion it forms part of Fallot's tetralogy and of Eisenmenger's syndrome The ventricular septal defect is usually at the upper part of the septum, involving the pars membranacea septi In this situation it may or may not be associated with a congenital defect of the auriculo-ventricular bundle; that is to say, with a congenital heart block

V S D. is commonly thought to produce a thrill and loud murmur at the sternal edge of the 4th left space, and to be consistent with long life, efficient cardiac action, and absence of cyanosis or breathlessness, but liability to bacterial endo-

A

FIG [illegible]

A Antero-posterior view Note the globular heart shadow, absent aortic knob, and dilated pulmonary artery branches

carditis Wood maintains that such cases are rare, many of those formerly diagnosed as V S D having had [illegible] murmurs [illegible] functional

with or without a thrill over the pulmonary artery and occasionally as low as the 4th space. Radiologically the pulmonary artery and its main branches are much dilated, the left ventricle is much enlarged and the left auricle is slightly enlarged.

B

FIG. 84.—Atrial and ventricular septal defects with complete congenital block (*contd.*)

B. *Right oblique view.* Note slight left auricular enlargement.

Cardiograms often show partial right bundles-branch-block.

A V.S.D. (like an A.S.D.) may become the seat of bacterial endocarditis. Emboli are usually carried into the right ventricle and lodge in the lungs; the symptoms are fever with

recurring lung infarcts. Less often, there are systemic emboli. Treatment is that of the complicating endocarditis.

FIG 84 —Atrial and ventricular septal defects with complete congenital block (contd)

C Left oblique view Note enlargement of left ventricular contour

3. Patent Ductus Arteriosus

Failure of the ductus arteriosus to close at birth (as it should do) is by no means uncommon It may be the only lesion, or may complicate other anomalies as already noted. As an isolated lesion it is compatible with total absence of subjective symptoms or it may give rise to slight breathlessness and cyanosis In the course of time subacute bacterial endocarditis may appear as a complication, or left ventricular failure may

develop. Some patients fail to grow, remaining small and underdeveloped.

The characteristic murmur may be the only abnormal physical sign; it is a continuous murmur, usually loud and harsh during *systole, and becoming softer during diastole*; *it* is best heard in the second left interspace about half an inch or an inch from the sternal border. The murmur is sometimes audible in the back, just to the left of the fourth or fifth thoracic spine, and rarely it can be heard here but not anteriorly. I

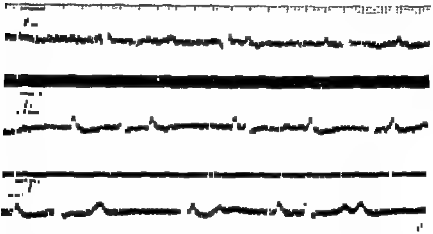


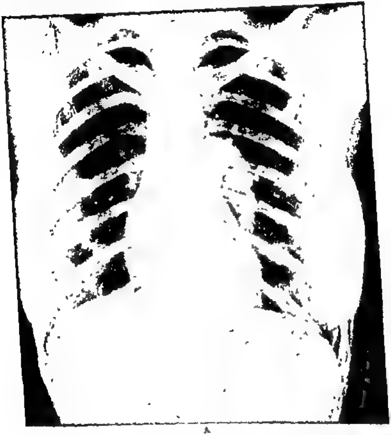
FIG 84 —Atrial and ventricular septal defects with complete congenital block (*contd*)

D *Cardiogram showing complete auriculo-ventricular block* The auricular complexes are normal The ventricular complexes resemble those seen in dextrocardia, the entire complex being inverted in lead I

have on one occasion encountered a patent ductus arteriosus in association with a right-sided aortic arch, the murmur was right-sided but otherwise characteristic The murmur is strongly suggestive of the diagnosis, rarely a similar murmur occurs with a septal defect or with a cavernous angioma If the ductus is widely patent, the pulmonary artery may become dilated. This may be recognised radiologically, and occasionally it is of sufficient degree to produce dullness on percussion to the left of the sternum in the second interspace (Gerhardt's sign) Occasionally aneurysmal dilatation of a patent ductus occurs

A widely patent ductus has an effect on the blood pressure similar to that of an arterio-venous aneurysm or of aortic

regurgitation; that is to say, the diastolic pressure falls while the systolic remains normal or rises. If the degree of patency is slight, exercise may be needed to demonstrate this char-



A

FIG. 85.—Patent ductus arteriosus. Female, aged 21. Murmur found on routine examination at age of 5. No breathlessness, but occasional [illegible]

A. Antero-posterior view, showing enlargement of the pulmonary conus and of the left branch of the pulmonary artery.

acteristic. Bohn's exercise test consists in noting the resting blood pressure, then recording the pressure immediately after simple exercise, even ten "knee bends" being sufficient,

readings should be repeated, more than once within the first minute if possible. The characteristic feature is a drop in the diastolic pressure, often nearly to zero, and its prompt recovery

B

FIG. 83 —Patent ductus arteriosus (*cont'd*)

B. *Right oblique view* showing absence of enlargement of left auricle, slight downward displacement of the left main bronchus, and a prominent aortic impression from a left aortic arch

within a minute or two The systolic pressure and pulse rate may rise.

The entry of additional blood into the pulmonary artery results in an increased venous return to the left auricle and left ventricle, which in due course, become hypertrophied The elevated pressure in the pulmonary artery leads also to hypertrophy of the right ventricle. If failure develops, it is

usually left ventricular but sometimes combined. A patent ductus sometimes becomes the seat of bacterial endocarditis,

C

FIG. 85.—Patent ductus arteriosus (*contd.*).

C *Left oblique view*, showing left aortic arch. On screening, the dilated left pulmonary artery was visible in the aortic window, but on the film this has been obscured by the barium-filled oesophagus.

in which case the characteristic murmur is accompanied by fever and recurring pulmonary infarcts.

Diagnosis.—The murmur of a patent duct may be simulated by an arterio-venous aneurysm, a cavernous angioma at the hilum of the lung, or rarely by an atrial or ventricular septal defect. I have known operation performed on one patient with

cavernous angioma and one with a septal defect. Neither condition produces the characteristic blood-pressure changes found with a patent duct. On the other hand, *the murmur in patent duct may be systolic only*, in which case the diagnosis is apt to be missed; it should be considered as a possibility *in all cases with a pulmonary systolic murmur and thrill*. In doubtful cases cardiac catheterisation, kymography (Shirley Smith), tomography (Tiscenco), and possibly angiocardiography may prove decisive.

Treatment.—Patent ductus arteriosus is compatible with survival to adult life, and cases have been known to survive to old age. A case has been recorded in which a patent ductus is thought to have closed spontaneously at the age of 20. Recently surgical ligation has been much practised. The chief difficulty in assessing the value of this procedure lies in the fact that there is no general agreement regarding the prognosis in *patent ductus arteriosus*; *the frequency of spontaneous closure* and of complications such as bacterial endocarditis or heart failure are unknown. Operation is not invariably successful in producing closure, nor is it entirely devoid of risk. If the lesion is uncomplicated and is causing neither symptoms, retardation of growth, nor cardiac enlargement, it is probably best left alone; but a close watch should be kept on the size of the pulmonary artery and left ventricle, signs that either of these is beginning to enlarge justify operation. Operation should be advised if there is retardation of growth, breathlessness, or interference with the child's capacity to lead a normal life. Finally, operation is imperative when the duct is infected (*i.e. complicated by bacterial endocarditis*), *prior to* the advent of penicillin, it offered the only hope of cure, chemotherapy with sulphonamides *is unnecessary and may result in waste of* valauble time. It is too early as yet to say whether penicillin will control the infection in these cases, but it seems likely that operation will be desirable nevertheless. The optimum age for operation is between the ages of 5 and 10. Ligation is clearly contra-indicated when there are additional malformations such as pulmonary *stenosis*, *these will be suggested* by the presence of significant cyanosis. Concomitant coarctation of the aorta is an absolute bar to ligation of a patent duct. Mitral stenosis renders ligation a procedure of questionable value.

4. Eisenmenger's Complex

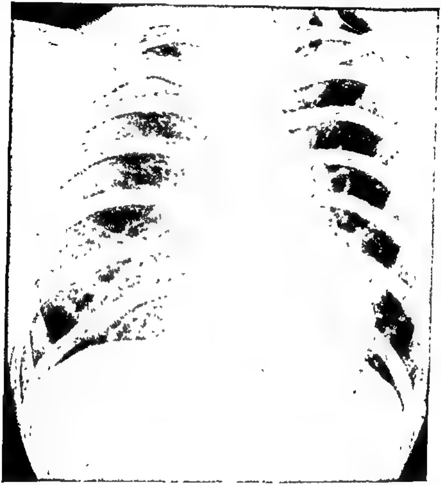
In Eisenmenger's complex there is a high ventricular septal defect with slight dextroposition of the aorta By dextroposition of the aorta is meant that the aorta overrides the septal defect, so that it arises partly from the right and partly from the left ventricle The pulmonary artery in the Eisenmenger complex is either normal in size or enlarged. Most of the blood from the right ventricle enters the pulmonary artery ; a small amount, insufficient to cause visible cyanosis, enters the aorta Cyanosis is usually absent at birth . it may remain absent throughout life or may develop during childhood in consequence of secondary changes in the lungs which increase the volume of the shunt. In the pre-cyanotic stage a loud systolic murmur and thrill may be present in the pulmonary area, often accompanied by signs of aortic regurgitation since the malformation is frequently accompanied by anomalies of the aortic cusps. X-ray shows enlargement of the left ventricle . the pulmonary artery may be normal or enlarged. In the cyanotic stage the clinical features may suggest Fallot's tetralogy or pulmonary stenosis , but in these conditions cyanosis and clubbing are present from birth and the pulmonary artery is small radiologically. The distinction is important, for Eisenmenger's complex is not as yet amenable to surgical treatment

THE CYANOTIC GROUP (*MORBUS COERULEUS*)

5 Congenital Pulmonary Stenosis, Congenital Pulmonary Atresia and Fallot's Tetralogy

The stenosis may affect the valve orifice, the cusps of which are fused to form a diaphragm , it may occur about an inch proximal to the site of the pulmonary valve which itself remains normal (infundibular stenosis) , finally, the pulmonary artery itself, or one of its main branches, may be narrowed or obliterated When the orifice is merely narrowed, the condition is referred to as " stenosis " , when completely obliterated, as " atresia " Pulmonary atresia, or pulmonary stenosis of any considerable degree, is incompatible with life unless there is a septal defect, either atrial or ventricular. Milder degrees of pulmonary stenosis are compatible with survival , but in most

cases in which birth is survived, a septal defect accompanies the pulmonary lesion, and the ductus arteriosus often remains patent as well, a partial right-to-left shunt occurs through



A

FIG. 86 – Pulmonary (infundibular) stenosis with ventricular septal defect and patent ductus arteriosus Male, aged 23, with intense cyanosis and clubbed fingers The continuous murmur of a patent ductus arteriosus was present as well as a loud systolic murmur, maximal at the sternal end of the 4th left costal cartilage

A *Antero-posterior view*, showing marked prominence of pulmonary conus The aortic knob is small, almost invisible

the septal defect and a left to right shunt through the ductus arteriosus. *Fallot's tetralogy* consists of pulmonary stenosis, right ventricular hypertrophy, a ventricular septal defect, and an overriding dextroposed aorta

Cyanosis is intense and is present from birth; clubbing of the fingers develops early. Breathlessness is easily induced, and the capacity for effort is much impaired. Paroxysms of increased cyanosis and breathlessness may occur without

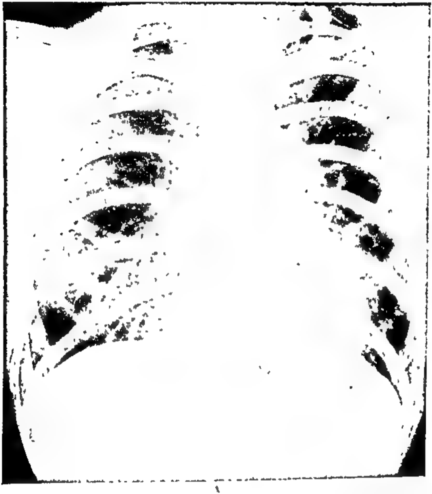
B

FIG. 80 —Pulmonary (infundibular) stenosis with ventricular septal defect and patent ductus arteriosus (*contd*)

B *Right oblique view* showing absence of [illegible] auricular enlargement There is a well marked aortic impression on the œsophagus from a left sided aortic arch (See also Figs 80, C and 80, D)

apparent cause The dyspnoea and cyanosis often lead the victim to adopt a "squatting" attitude The physical findings, in addition to the cyanosis and clubbed fingers, are enlargement of the right ventricle with epigastric pulsation, and a palpable thrill with a coarse systolic murmur to the left of

cases in which birth is survived, a septal defect accompanies the pulmonary lesion, and the ductus arteriosus often remains patent as well; a partial right-to-left shunt occurs through



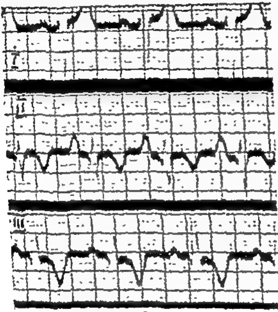
A

FIG. 80.—Pulmonary (infundibular) stenosis with ventricular septal defect [illegible] 23, with intense cyanosis and [illegible] of a patent ductus arteriosus [illegible] murmur, maximal at the sternal end of the 4th left costal cartilage.

A. *Antero-posterior view*, showing marked prominence of pulmonary conus. The aortic knob is small, almost invisible.

the septal defect, and a left-to-right shunt through the ductus arteriosus. *Fallot's tetralogy* consists of pulmonary stenosis, right ventricular hypertrophy, a ventricular septal defect, and an overriding dextroposed aorta.

ventricle, the latter forms the apex of the heart while the left ventricle remains small: the pulmonary conus and pulmonary artery also remain small. The heart shadow acquires a characteristic shape with a concavity in the position of the pulmonary artery and a prominent, raised apex (*cœur en sabot*). The pulmonary vascular shadows are diminished In a small



D

FIG. 80 —*Pulmonary (infundibular) stenosis with ventricular septal defect and patent ductus arteriosus (contd)*

D *Cardiogram from same case, showing large P waves in lead 2, and gross right axial deviation with inversion of T2 and T3 indicating right ventricular hypertrophy*

number of cases of pure pulmonary stenosis in whom the septal defect is auricular the right ventricle remains small, and the cardiogram shows left axial deviation

The victims of pulmonary stenosis are unusually susceptible to infections and usually die from an intercurrent infection during childhood or adolescence It is rare for them to pass the age of 20

Treatment.—Surgical treatment is now possible for some of these cases In pure pulmonary valvular stenosis and in isolated infundibular stenosis, Brock has successfully excised

the sternum in the third, or second and third interspaces. In the presence of the pulmonary stenotic murmur it is rarely possible to recognise the murmur of a concomitant septal defect, which must be assumed rather than diagnosed; it may be

C

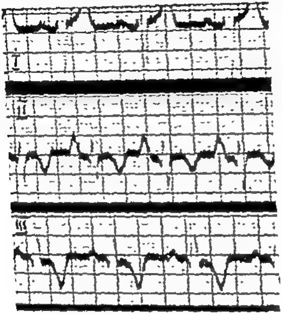
FIG 86.—Pulmonary (infundibular) stenosis with ventricular septal defect and patent ductus arteriosus (*contd*)

C *Left oblique view*, showing aortic arch and absence of enlargement of ventricular contour

possible to recognise the continuous murmur of a concomitant patent ductus arteriosus as the stethoscope is moved farther away from the sternum In the case of pulmonary atresia, of course, the pulmonary stenotic thrill and murmur are absent.

In the usual cases with ventricular septal defect, cardiograms show gross right axial deviation with broadening of QRS and inversion of T in leads 2, 3 and the right-sided chest leads. X-ray shows enlargement of the right auricle and right

ventricle; the latter forms the apex of the heart while the left ventricle remains small: the pulmonary conus and pulmonary artery also remain small. The heart shadow acquires a characteristic shape with a concavity in the position of the pulmonary artery and a prominent, raised apex (*cœur en sabot*). The pulmonary vascular shadows are diminished. In a small



D

FIG. 86.—Pulmonary (infundibular) stenosis with ventricular septal defect and patent ductus arteriosus (*contd.*)

D. *Cardiogram from same case, showing large P waves in lead 2, and gross right axial deviation with inversion of T2 and T3 indicating right ventricular hypertrophy.*

number of cases of pure pulmonary stenosis in whom the septal defect is auricular the right ventricle remains small; and the cardiogram shows left axial deviation.

The victims of pulmonary stenosis are unusually susceptible to infections and usually die from an intercurrent infection during childhood or adolescence. *It is rare for them to pass the age of 20.*

Treatment.—Surgical treatment is now possible for some of these cases. In pure pulmonary valvular stenosis and in isolated infundibular stenosis, Brock has successfully excised

the stenosed portion, approaching it through the right ventricle; the technique is difficult and the mortality is high, but if successful the procedure is apparently curative. In the more frequent cases of Fallot's tetralogy, palliative operations are used with striking benefit. The aim is to provide an additional blood supply to the lungs by creating an "artificial ductus". This may be done by anastomosing a systemic artery (usually the subclavian) to one of the pulmonary artery branches (Blalock-Taussig operation), or by anastomosing the aorta directly to the pulmonary artery (Pott's operation). Feasibility of the operations depends on the anatomical situation, size, and patency of the pulmonary artery branches, which must be assessed beforehand either by fluoroscopy or by angiocardiography. In occasional cases one or other branch of the pulmonary artery is completely occluded so as to render surgical anastomosis impracticable. Cases with radiological dilatation of the pulmonary artery are generally unsuitable. When practicable, the operation is followed by abolition or great reduction of the cyanosis and by striking improvement in the capacity for effort. It is too early to assess the ultimate outlook; left ventricular hypertrophy, as in spontaneous patency of the duct, would seem to be a likely sequel though less incapacitating and less dangerous than the original malady.

6. Tricuspid Stenosis or Atresia

Tricuspid atresia is associated with defective development of the right ventricle; this chamber may be absent, it may take the form of a blind sac, or it may be small receiving blood from the left ventricle through a septal defect. The pulmonary artery is small or occluded. Venous blood enters the left auricle through an auricular septal defect; the lungs receive their blood supply through a patent ductus arteriosus, and the oxygenated blood returning to the left auricle is mixed with venous blood.

Cyanosis and clubbing are present from birth and are intense. The liver is enlarged and there is pre-systolic hepatic pulsation. The shadow of the pulmonary conus is absent on X-ray examination, giving a characteristic shape to the heart shadow in all three views. Cardiograms show left axial devia-

tion The Blalock-Taussig operation may prove of value in these cases

7 Malformations of the Aorta and Pulmonary Artery

The aorta and pulmonary artery are developed from a single vessel, the *truncus arteriosus*. A spiral septum divides the truncus into two vessels. Normally the spiral septum meets the interventricular septum and fuses with it in such a manner that the pulmonary artery is joined to the right ventricle and the aorta to the left ventricle. Failure of the spiral septum to develop results in persistence of a common trunk into which both ventricles open. Failure of the spiral septum to meet the interventricular septum at the correct angle leads to complete or partial transposition of the great vessels

Persistent Truncus Arteriosus.—Both ventricles open into a common trunk from which innominate, left common carotid, left subclavian and descending thoracic aorta arise . In some instances right and left pulmonary arteries also arise from the common trunk in these cases an adequate volume of blood is oxygenated and cyanosis is slight or absent *In other* instances the pulmonary arteries are atresic the lungs receiving their sole supply from the bronchial arteries in these circumstances cyanosis is intense There is usually a basal *systolic* murmur which is not diagnostic Diagnosis depends on radiological evidence supported by angiocardiography and cardiac catheterisation

Complete Transposition of the Great Vessels.—The aorta rises from the right ventricle receiving venous blood which is distributed throughout the body and returned to the right auricle The pulmonary artery rises from the left ventricle and receives arterial blood which is passed through the lungs and returned to the left auricle Unless a shunt persists after birth the condition is incompatible with extra-uterine life A shunt may persist through an auricular septal defect, a ventricular septal defect, or a patent duct When the shunt is through a septal defect cyanosis is intense and uniform both ventricles undergo great enlargement When the shunt is through a patent duct, oxygenated blood escapes from the pulmonary artery into the descending aorta and is distributed

to the legs; cyanosis is intense in the head and trunk but less intense in the legs, and a definite line of demarcation is visible at the brim of the pelvis. In these cases the murmur of the *patent duct may be audible. Complete transposition of the* great vessels is usually only compatible with life for a few months.

Partial Transposition—Dextroposition of the Aorta.—Varying grades of transposition are met with. The aorta may override a septal defect arising partly from the left and partly from the right ventricle. Similarly, the pulmonary artery may arise in part from the right and in part from the left ventricle. Dextroposition of the aorta occurs as an integral part of Fallot's tetralogy, and slight dextroposition characterises Eisenmenger's complex. Dr Helen Taussig has recently differentiated a new syndrome, hitherto confused on the one hand with Fallot's, and on the other with Eisenmenger's malformations.

Taussig's Syndrome.—There is a high septal defect with an overriding pulmonary artery and a transposed aorta, the pulmonary artery arises from both ventricles, the aorta from the right ventricle only. The victims of Taussig's syndrome resemble Fallot's tetralogy in that cyanosis is present from birth, but in all other respects they resemble Eisenmenger's complex. The following quotation is from Taussig's abstract.

> Clinically it resembles the Eisenmenger complex in many features Both are compatible with life for a number of years In both, the heart is slightly, if at all, enlarged Contours of the hearts are similar in that both show fullness of the pulmonary artery and hilar pulsations. Neither is characterised by squatting Both show cardiographic evidence of right ventricular hypertrophy This malformation differs from Eisenmenger's complex in that cyanosis dates from birth whereas late development of cyanosis is characteristic of the Eisenmenger complex

The condition is not amenable to surgical treatment.

Taussig's Criteria for the Blalock-Taussig Operation.—*Taussig gives the following criteria for the* Blalock-Taussig operation, analogous considerations are applicable to the Pott's operation

(1) The primary difficulty must be lack of adequate circulation to the lungs.

(2) The size and structure of the heart must be such that it can adjust to the altered circulation

(3) There must be a systemic artery of suitable size and length to use for the anastomosis.

(4) There must be a pulmonary artery to which to anastomose the systemic artery; furthermore, both the structure of, and the circulation to, the opposite lung must be such that the patient can survive the temporary occlusion of one pulmonary artery during the time required for the anastomosis.

(5) The relative pressure in the two circulations must be such that blood will flow through the new pathway to the lungs

THE ACYANOTIC GROUP

8 Coarctation of the Aorta

Although six different types of coarctation have been described by Evans, they fall into two main groups, namely those with and those without a patent ductus arteriosus

Coarctation without Patency of the Ductus.—There may be a localised stricture at the level of the ligamentum arteriosum, or there may be complete atresia of a segment of the aorta above this level In consequence, hypertension develops in the ascending aorta, arms, and head, this is followed by hypertrophy of the left ventricle Contrasting with the high pressure in the arms is the low pressure in the descending aorta and legs A collateral circulation develops, involving anteriorly the internal mammary with the superficial epigastric arteries, and posteriorly the scapular and intercostal vessels.

The patients are apt to complain of hypertensive cerebral symptoms such as headaches or tinnitus Subarachnoid haemorrhage is a relatively frequent complication and is often the cause of death in those under 30. Alternatively, there may be anginal pain or breathlessness with ultimate development of left ventricular failure The diagnosis should be suspected when hypertension is found *in a* young person who has not suffered from kidney disease and whose family history is negative It is confirmed by the pressure difference between the arms and legs and by the collateral circulation. Arteries may be felt in abnormal situations in the neck, pulsation of the enlarged internal mammary and superficial epigastric

arteries can sometimes be felt; enlarged arteries in the back, especially in the interscapular space, are best shown by placing the patient prone with a pillow under his chest and allowing

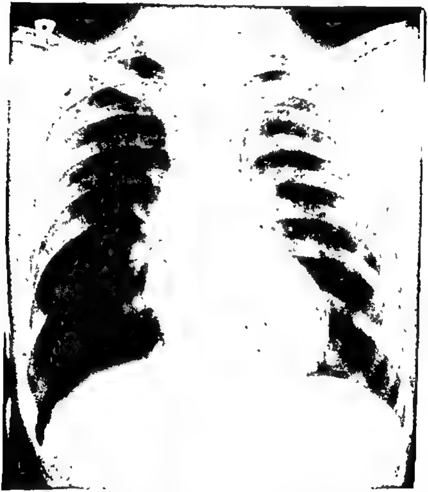


FIG. 87—Rib-notching from coarctation of aorta Note the lower borders of the posterior aspects of the 9th and 10th ribs on each side Bramwell's sign (double aortic knob) is also visible, and the left ventricular contour is prominent

his arms to hang down from the sides of the couch or bed. The dilated intercostal vessels usually erode the lower borders of the ribs, producing the characteristic radiological sign known as "rib-notching". Rarely, the actual stricture can be visualised on the fluorescent screen in the left oblique position The hypertension in coarctation develops between the ages of

7 and 15, the systolic pressure rising from an average of 140 to an average of 180 during this period (Campbell); occasionally the pressure in the arms is not raised even in adults (Bramwell). The chief danger period in coarctation is the third decade; once the age of 30 has been passed the prognosis is reasonably good.

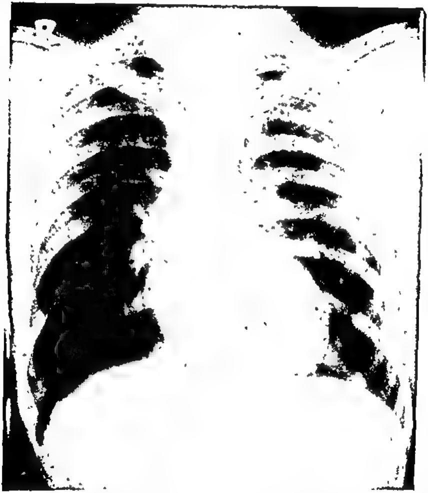
Coarctation with Patent Ductus.—The circulation to the lower extremities is largely supplied through the patent duct which communicates with the aorta below the stricture The blood flow to the lungs is less, the venous return to the left side of the heart is correspondingly reduced. The rise of pressure in the arms is smaller while the pressure in the legs is not so low, the pressure difference being less obvious. Left ventricular hypertrophy may be absent. The superficial anastomotic channels do not develop, or do so only to a slight extent; and the radiological sign of rib-notching is usually absent. While the murmur of the patent duct can be recognised, the coarctation is apt to be overlooked. Bramwell describes a characteristic radiological sign in this type of case, viz a double aortic knuckle in the postero-anterior view; the upper projection represents the blind end of the aortic arch while the lower projection is formed by the upper end of the descending aorta The prognosis is better with patency of the duct as the risk of subarachnoid haemorrhage or of left ventricular failure is less

Treatment.—A number of cases have now been successfully treated by excision of the constricted segment of the aorta with end-to-end anastomosis The practicability of operation is governed by the length of the constricted segment Cases with patent ductus are unsuitable for operation When operation is impracticable, treatment is symptomatic along lines similar to those employed in essential hypertension

9. Sub-aortic Stenosis

In some cases there is congenital stenosis of the aortic valve In sub-aortic stenosis there is a stricture by a band of fibrous tissue or membrane which stretches across the orifice about one centimetre below the aortic valve The lesions cause gross hypertrophy of the left ventricle; the blood and pulse pressures remain low. There is no cyanosis, but rather pallor.

arteries can sometimes be felt ; enlarged arteries in the back, especially in the interscapular space, are best shown by placing the patient prone with a pillow under his chest and allowing



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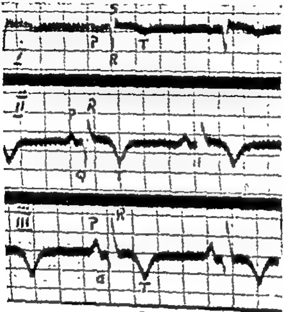
his arms to hang down from the sides of the couch or bed. The dilated intercostal vessels usually erode the lower borders of the ribs, producing the characteristic radiological sign known as "rib-notching". Rarely the actual stricture can be visualised on the fluorescent screen in the left oblique position The hypertension in coarctation develops between the ages of

congenital cardiac lesions. Uncomplicated dextrocardia is compatible with perfect health and long life. The apex impulse is found in the fifth right interspace instead of the left; the sounds are normal. The X-ray picture is a mirror image of the normal—it can be reproduced by reversing a normal X-ray film so that it is viewed from the back. The cardiogram



B

FIG. 89.—Dextrocardia (contd.)

is characteristic, all deflections in lead 1 are inverted, leads 2 and 3 are transposed. If, in a case of suspected dextrocardia, the electrocardiogram fails to show the characteristic features, the condition is probably a displacement of the heart, not a true dextrocardia. I have seen diaphragmatic hernia cause such a displacement as to simulate dextrocardia, but the characteristic cardiographic findings were absent; there was merely right axial deviation. In one case of dextrocardia, the occurrence of a coronary thrombosis during the fifth decade led to a bizarre

The physical signs are the same as those of acquired aortic stenosis. The prognosis is bad; subacute bacterial endocarditis and cardiac failure are the main dangers. No surgical treatment has yet been devised.

Bicuspid aortic valve cannot be diagnosed clinically as it produces no signs or symptoms. It is more liable to subacute

A

FIG 88—Dextrocardia (Dr A Glen's case) Male, aged 45, admitted on account of coronary thrombosis There was no history of previous cardiac symptoms, the attack was typical except for the fact that the pain was right-sided

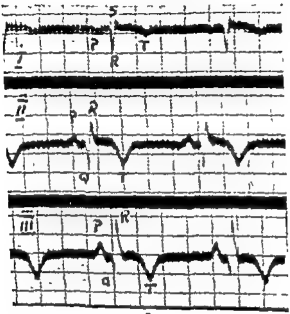
A *Teleradiogram showing dextrocardia* The measurements of the heart shadow are within normal limits, but the apex appears a little prominent

bacterial endocarditis than a normal valve, and may be suspected when bacterial endocarditis develops in an aortic valve which has hitherto seemed healthy.

10. DEXTROCARDIA

In this condition the heart is a mirror image of the normal and the aortic arch curves over to the right. Dextrocardia may be an isolated finding, it may be associated with transposition of the abdominal viscera, or it may accompany other

congenital cardiac lesions. Uncomplicated dextrocardia is compatible with perfect health and long life. The apex impulse is found in the fifth right interspace instead of the left; the sounds are normal. The X-ray picture is a mirror image of the normal—it can be reproduced by reversing a normal X-ray film so that it is viewed from the back. The cardiogram



B

FIG. 88.—Dextrocardia (contd.)

is characteristic, all deflections in lead 1 are inverted, leads 2 and 3 are transposed. If, in a case of suspected dextrocardia, the electrocardiogram fails to show the characteristic features, the condition is probably a displacement of the heart, not a true dextrocardia. I have seen diaphragmatic hernia cause such a displacement as to simulate dextrocardia, but the characteristic cardiographic findings were absent, there was merely right axial deviation. In one case of dextrocardia, the occurrence of a coronary thrombosis during the fifth decade led to a bizarre

cardiogram in which the characteristic inversion of lead 1 wa associated with coronary Q waves and coronary T waves i leads 2 and 3 (Fig. 88). Uncomplicated dextrocardia require no treatment.

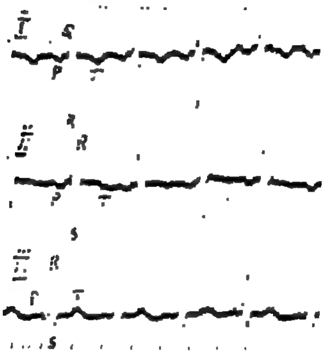


FIG. 89.—Cardiogram from a case of dextrocardia. Girl, aged 14; symptom free; condition found on routine school medical examination. Investigation showed complete transposition of all viscera—dextrocardia, *right aortic arch, right sided stomach, left-sided liver. The electrocardiogram shows inversion of all waves in lead 1; leads 2 and 3 are transposed; P2 is inverted in this record.* NOTE.—*The time markings in this cardiogram correspond to quarters and twentieths of a second.*

11. CONGENITAL HEART BLOCK

This is due to congenital absence of a portion of the auriculo-ventricular bundle; the block is therefore always complete. It may be associated with a ventricular septal defect, or may occur without this. The heart rate is rather higher than in acquired complete block, usually about 40 to 45. The condition is compatible with long life and comparatively little disturbance of the circulation. One of my patients, now aged 44, was found to have a slow pulse at school; he was told not to play

He next came under medical observation at the age of 30 when he developed influenza during an epidemic and his slow pulse was again noted; when he first returned to work after this he had a syncopal attack and was referred to me. He had a ventricular septal defect with complete congenital block. He has since worked continuously as a joiner, and during the war years had been doing additional work to compensate for the loss of one of his employees. He has had no further cardiac symptoms in the interval, though he has been seen at regular intervals by his medical practitioner in the course of visits to members of his family. On the other hand, patients with congenital complete block may suffer from Stokes-Adams attacks. Alternatively, if the block is associated with other congenital anomalies they may have symptoms due to the latter. A woman with complete congenital block, complicated by ventricular and atrial septal defects, suffered from cyanosis and breathlessness with occasional attacks of dizziness from her early schooldays; despite a breakdown at the age of 38 she continued to work as a housekeeper, though taking periodic rests while at work, till the age of 47 when she died suddenly. This case was fully reported a year before her death (Fig. 84, A to D).

Association of Congenital Cardiac Lesions with Other Congenital Defects

Congenital heart lesions are not infrequently associated with other congenital defects. Mongolism is frequently complicated [illegible] mo-

The congenital abnormalities above described are the common ones. The reader is referred to the writings of Maude Abbott for an anatomical and embryological description, and to Helen Taussig's *Congenital Malformations of the Heart* for a treatise on the clinical features and diagnosis.

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CHAPTER 20

THE CARDIOVASCULAR SYSTEM IN PSYCHONEUROSIS

PRECORDIAL PAIN OF NERVOUS ORIGIN

ATTACKS of precordial pain are very frequent in persons who are suffering from anxiety, especially if the anxiety is about their heart. Precordial pain may also occur as a manifestation of hysteria. Formerly this pain was known as "pseudo-angina" or "false angina", but nowadays there is no general name for it. "angina innocens" suggested by Dr. Geoffrey Bourne, is open to the same objections as pseudo-angina, namely that it suggests some sort of relationship to true angina. "False angina" is less objectionable, but I prefer to avoid the word angina, speaking of psychogenic or psychoneurotic pain.

Many of the patients with nervous precordial pain have normal healthy hearts and normal circulatory systems. But it should be noted that the presence of an organic heart lesion does not confer immunity against anxiety, and that patients with organic heart disease may become anxious and develop pain due to their anxiety as opposed to their cardiac lesion. In fact, patients with coronary disease frequently become anxious and develop anxiety pain in addition to their genuine coronary pain; the anxiety pain may be the more incapacitating of the two, but it can be cured by appropriate treatment, leaving only the genuine coronary pains.

The most important differentiating features of nervous pain lie in its behaviour. Patients will say that it comes on at any time, but especially in relation to fatigue or to excitement. Thus it is more apt to come on *after* effort than *during* effort, and if it should come on during effort, it rarely subsides when the effort ceases, but commonly persists for an hour or two, or for the remainder of the day. Apart from attacks during or after effort, attacks frequently occur while at rest. And conversely, effort does not always induce the attacks; often the patient has "good days" and "bad days"; on a good

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pain is not due to the heart condition but to nervousness; that his heart is not failing and is compatible with a number of years of useful and comfortable life.

In many cases an explanation on the foregoing lines will succeed in abolishing the pain without any other treatment. If there is insomnia, this should be corrected; 15 grains of ammonium bromide with 10 minims of liquid extract of ergot and 10 minims of aromatic spirit of ammonia given three or four times daily is an effective sedative for this purpose; the ergot appears to enhance the value of the ammonium bromide, while the aromatic spirit of ammonia lessens the likelihood of bromism. Alternatively, ½ grain of phenobarbitone may be given thrice daily.

On no account should a patient with a healthy heart be put to bed or restricted in any way for his allegedly cardiac pains; to do so merely annuls the effect of the positive statement that his heart is healthy. In some cases with considerable nervous tremor and excitement, it may be necessary to order rest in bed for the nervous state; it should be made clear to the patient that the rest is required for the disordered state of his nerves and not for his heart. In all other cases, work and exercise should be encouraged. Where the neurosis complicates organic heart disease, restrictions will be necessary; their extent is determined by the state of the heart, leaving out of account the psychoneurotic pains; and an appropriate explanation must be given.

Effort Syndrome

Definition.—A condition in which symptoms of circulatory inefficiency are present in the absence of any organic cause. The heart, arteries, and lungs are healthy; there is no anaemia and no thyrotoxicosis. The condition was formerly known as "disordered action of the heart", "soldier's heart", "irritable heart", "strained heart"; these are bad names because they suggest some obscure heart disease. It is now known as "effort syndrome" or "Da Costa's syndrome".

Nature and Causation.—It is now generally agreed that most, if not all, cases are suffering from an anxiety neurosis, of which the effort syndrome is a manifestation.

Just as exercise is necessary to maintain tone in voluntary

day he or she can take plenty of exercise (perhaps playing tennis, or several rounds of golf) without any pain whatever; but on "bad days" he will say that "he becomes exhausted and pain develops on the slightest effort". True angina never shows these gross variations in the behaviour from day to day; there may be variations attributable to temperature, wind, etc., and there may be improvement or deterioration over a long period.

The pain is more apt to be located to the cardiac apex than to the mid line. It may be a momentary stabbing pain, it may take the form of a whole series of such pains, or it may be described as constant, and lasting anything up to a whole day. It quite often radiates to the left arm, but in a very large series of cases I have never seen it go to any of the other areas to which genuine anginal pain sometimes spreads.

Occasionally, pain behaving as described above is the sole evidence of an anxiety state—the patients seem placid enough and show no other abnormality, enquiry, however, will usually elicit the information that they are off their sleep, and that this symptom preceded or coincided with the appearance of the pain Further enquiry may elicit a precipitating factor —onset following a sudden cardiac death in the family, following the discovery of a hitherto silent cardiac lesion at routine examination, or even following the accidental discovery (e g. during examination for a febrile cold) of an innocent murmur which is misinterpreted More often, however, the existence of the anxiety state is obvious the patient is tense, nervous, and anxious, he has sweaty palms and axillae, with tremor of tongue and fingers, tachc. and exaggerated reflexes.

Treatment.—The first step in treatment consists in stating positively that the patient has a healthy heart, it is not sufficient to say that "there is no evidence of heart disease" or "I cannot find anything wrong". Next, the patient must be given an explanation of his pain, the exact approach will depend on his intelligence and insight, in some cases a statement that the pain is a "nervous pain" or that it is "due to anxiety" will suffice, but other patients will require a more detailed explanation. In the case of anxiety which complicates an organic heart lesion a slightly different course is necessary. The patient is told that he (or she) has a specified lesion, but that it is not as bad as he has been led to believe, that his

development. But it is a great mistake to think that it is a disease peculiar to war-time or to the Army; it is quite common in civilians and in peace-time. It is more common in the Army than in the Air Force or Navy, probably because of differences in the nature of the effort demanded—e.g. long marches with heavy equipment are more likely to elicit the symptoms than a shorter though more severe effort.

Symptoms.—The chief symptom is breathlessness on exertion, but it is rare for this to be the only symptom. Insomnia or disturbed sleep is present in nearly every case and usually right from the start. Palpitation on exertion or on excitement is almost as common; sometimes there are attacks of extrasystoles or paroxysmal tachycardia of which the patient is aware. The remaining symptoms are less constant, though still frequent. Some patients complain of pain, usually located to the cardiac apex, but occasionally radiating to the left arm, coming on when excited or after effort; less often the pain is more like a true angina developing during effort, though as a rule it is not rapidly relieved by rest. There is a tendency to sweat on slight effort. Faintness, dizziness, or actual fainting attacks are frequent. Some of the patients suffer from dyspeptic symptoms as well.

Physical Signs.—In the majority of cases the patient is well nourished and of good colour, but occasionally he is poorly nourished, debilitated, or of poor physique. Many are obviously nervous, though some do not show it. The hands are frequently cold, congested, and blue. The palms and axillae

e beads

Some

patients exhibit a tendency to flush; in women especially, an erythematous morbilliform rash may appear over the front of the chest, fading a few minutes later.

Pulse. The findings are variable. In some cases the pulse is normal and gives a normal response to exercise, despite which the latter may cause breathlessness; if so, the respirations tend to be shallow though rapid. Frequently there is a normal resting pulse rate, but an excessive rise in response to effort or delay in the return to the resting value afterwards. Some patients with effort syndrome show a considerable difference in pulse rates taken seated and standing. In another group there is resting tachycardia, with normal or impaired

muscles, it is required to maintain the tone of the circulation. Persons confined to bed for any reason, and those who lead a sedentary life, soon get "out of training"; when they begin to exercise again they are short-winded at first and they may have palpitation. This condition may be described as *neuro-circulatory asthenia*, the causes of which are:

(1) Convalescence from acute illness
(2) Chronic infections—T.B., genito-urinary infections, etc.
(3) Sedentary habits
(4) Poor general physique due to malnutrition or other cause.

In healthy persons, the symptoms soon pass off with graduated exercise and training if due to convalescence or sedentary habits; if due to a chronic infection or malnutrition they pass off with graduated exercise provided the underlying cause is also rectified. But if an element of anxiety concerning his heart is kindled in the patient's mind, the symptoms persist and the fully fledged picture of *effort syndrome* develops. The anxiety may be induced by indiscreet remarks by doctor or nurse during an acute illness, e.g. that the heart has been "weakened", or that it has been "poisoned"; it may be due to an over-anxious mother or wife, either in the past or present; it may be due to contact with, or knowledge of friends or relatives who suffer from genuine heart disease; it may be due to impending court proceedings plus differing reports from many doctors in workmen's compensation or other accident cases; it often arises when a youth who has led a sheltered life watched over by anxious parents is exposed to the rigours of army life. Doctors and nurses are responsible for a good many cases, either by lack of tact, by insufficient firmness, or by "erring on the safe side". In some cases the symptoms follow directly on an acute illness such as influenza or sometimes rheumatic fever; some begin after an accident, particularly if this has involved injury to the chest wall. Alternatively, the symptoms may develop only after a lapse of years in response to some psychological difficulty; the seeds laid in youth lie dormant until awakened by fear, anxiety, or worry.

Effort syndrome is more common in males than females, though it occurs in both sexes; it occurs at all ages but is especially common in young adults; it becomes more prevalent in war-time because the conditions are more favourable for its

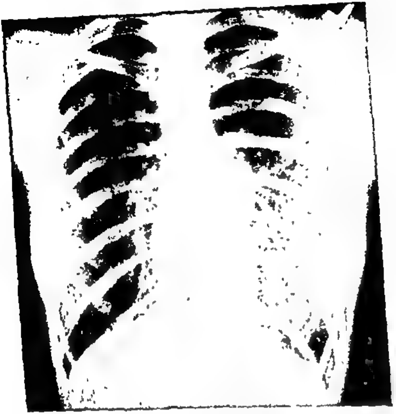


FIG 90—Small heart in neuro-circulatory asthenia Girl aged 13 Her doctor writes "She was under my care a year ago because of nervousness and poor sleep, at that time she had no cardiac symptoms but was growing very quickly Two months ago the school doctor sent her to me, her heart was galloping, irregular in rhythm, with a murmur at the apex, but I felt the condition was nervous rather than organic." The patient gave a history of one or two fainting attacks a year previously, but she had no breathlessness She was tall and thin, active, and had a good appetite The skin showed tache, but there was no thyroid enlargement Menstruation had started at 11½, and was regular There was tachycardia (pulse

but no evidence of

normal Note the

full sized film as used for adults), and the small vertical heart shadow as though the growth of the heart had failed to keep pace with that of the trunk The lungs are free from any sign of tuberculosis There ..

response to exercise but breathlessness in either case. In this group it will often be noted that after cessation of exercise the pulse rate drops within a minute to a normal figure; but that within another 15 to 30 seconds it rises again. This type of behaviour of the pulse in any patient with a tachycardia suggests that the latter is of nervous origin. It is best demonstrated by recording the number of beats in each successive period of ten seconds after termination of the exercise.

The *blood pressure* is sometimes normal, but more often the systolic pressure is raised and the pulse pressure high. In many cases the blood pressure is unstable; thus the initial reading may be 170/100 or even higher, but the figure gradually falls as repeated readings are taken; sometimes it ultimately reaches a normal value, or it may alternately fall and rise. In some patients there is a considerable difference between the blood pressure when seated and when lying; the difference is not in the same direction in every case.

Heart. All findings may be quite normal. More often *pulsation is diffuse and excited; this frequently leads to a* mistaken idea that the heart is enlarged; but careful location of the apex will show that it is not, while percussion confirms that the cardiac dullness is of normal extent. There is often tenderness over the position of the apex. X-ray examination shows a heart shadow of normal size; not infrequently the heart is smaller than average (Figs. 90 and 91). Individuals with unusually small hearts appear to be more prone to develop neuro-circulatory asthenia or effort syndrome. The heart sounds are usually loud, sometimes slapping; systolic murmurs at the apex or the base, or cardio-respiratory murmurs, may be present. The electrocardiogram is of normal type, sometimes showing a simple tachycardia, the P and T waves are often relatively high, while the PR interval is comparatively short. Extrasystoles or sinus arrhythmia may be recorded.

Central nervous system. Tremor of the tongue is almost always present. Tremor of the fingers is frequent, though less constant; it may be fine or coarse. The tendon jerks are often very active, being elicited by a very light tap and giving an exaggerated response.

Urine. Orthostatic albuminuria is frequently present in patients with effort syndrome, though the converse does not hold good.

be expected; *it is precisely* in these cases that it gives a "borderline" *figure*; the clinical diagnosis is rarely in doubt when the metabolic rate is either normal or significantly raised. It is questionable whether a diagnosis of thyrotoxicosis is ever justified in a patient with a normal pulse rate and normal blood

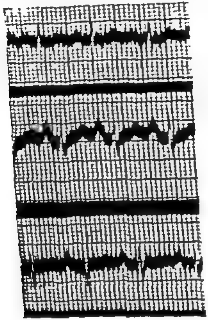
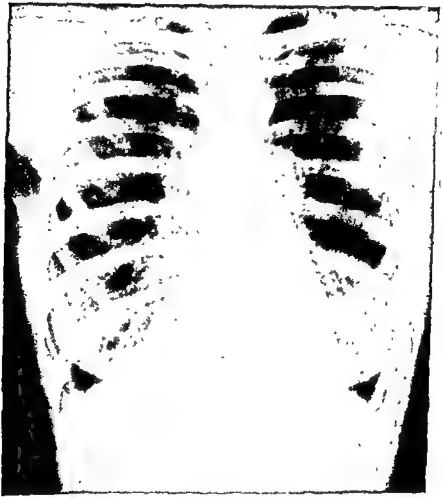


FIG. 91—Small heart in neuro-circulatory asthenia with effort syndrome [illegible]

ment and no eye signs of thyrotoxicosis, but tache present with tremor of tongue and fingers Heart sounds pure, pulse 120, BP 126 78, arteries healthy Cardiogram shows tremor, relatively tall P waves in lead 2, and normal ventricular complexes The X ray shows a small vertical heart shadow, in the lung fields there is slight increase in bronchial markings consistent with chronic bronchitis, but no sign of tuberculosis The patient improved and put on weight when reassured about her heart and treated with sedatives

pressure, whatever may be the figure given for the basal metabolic rate Absence of loss of weight does not exclude thyrotoxicosis, though it is unusual. *Neuro-circulatory asthenia* due to convalescence is easily diagnosed from the history. Before

Diagnosis.—The cardiac signs might simulate *endocarditis*; absence of cardiac enlargement and of diastolic murmurs, normal temperature and blood sedimentation rate, and presence of signs of anxiety in the shape of a tremulous tongue and



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moist palms, are important diagnostic features *Thyrotoxicosis* produces a very similar picture, and in some cases it is extremely difficult to distinguish from effort syndrome. Absence of thyroid enlargement, absence of loss of weight, and a normal sleeping pulse rate favour effort syndrome as the diagnosis. It is necessary to remember, however, that in thyrotoxicosis the thyroid is sometimes retrosternal and impalpable. The basal metabolic rate is of less assistance in doubtful cases than might

Having once confirmed the diagnosis, the patient must not only be told that he has a healthy heart, but must be induced to believe this. He should be told that he is temporarily out of training as a result of insufficient sleep or of nervousness, and that he will make a complete recovery as a result of his own efforts. If there is insomnia or much tremor, sedatives should be ordered, the bromide and ergot mixture recommended for precordial pain of nervous origin (p. 435) is effective. A course of graduated outdoor exercise should be prescribed and should be insisted upon, some patients are inclined to slack or to funk the exercise unless they are under supervision At an early stage the exercise should have reference to something useful (work) or to something interesting (games). This is the basis of "occupational therapy" as used in institutions, but with a little ingenuity it can be applied in the patient's own home An early return to work should always be advocated; prolonged periods on the sick list are demoralising.

In a high percentage of cases, fear of heart disease is the underlying cause of the patient's anxiety, and in these circumstances a complete recovery can readily be achieved by the simple treatment outlined above In a smaller number of patients there is some other additional cause of anxiety; for example, one of my patients, on being asked bluntly what source of anxiety he was concealing, admitted that, prior to his marriage, he had an illegitimate daughter whose existence was unknown to his wife, the daughter was "boarded out" and the landlady was blackmailing the patient. He was advised to make a clean breast of the matter to his wife; he wrote me a month later to say he had taken my advice, that his symptoms had all vanished, and that he was feeling well and back at work after nine months on the sick list labelled "myocarditis" Sometimes the patient's problem is less easily solved or it may be insoluble; even in such a case the fact of "getting it off his chest" usually leads to improvement There remain a small number of patients who appear to be suffering from some deep-seated anxiety for which no cause can be discovered, in these cases the help of a psychiatrist is required

labelling a case psychoneurotic, it is important to exclude chronic infections, especially tuberculosis and chronic pyuria, and to treat these conditions if present. *Emphysema* as a cause of breathlessness may be overlooked; though it should not be missed if the chest is inspected from the side.

Patients with organic heart disease sometimes develop a superimposed effort syndrome. This should be suspected if *complaint of breathlessness on exertion is out of proportion* to the severity of the cardiac lesion. The suspicion is confirmed by taking note of the circumstances in which the increased breathlessness first made its appearance, by enquiring about insomnia, and by seeking tremor of the tongue and sweating of the palms.

Prevention.—The seeds of effort syndrome are often sown *by an anxious mother in childhood, sometimes by an indiscreet* doctor or nurse. Unnecessary restrictions on the activities of a healthy child may well lead to a sense of inferiority, as may also excessive "coddling". During an acute illness in which the heart is not in fact damaged, it is important to avoid any suggestion that it might be damaged. If restrictions are imposed during convalescence, or if graduated exercise is prescribed in a patient with a normal heart, he should not be told that these are "to tone up his weak heart", but that they are "to get him back into training". In the case of an *accidentally discovered murmur*, immediate steps should be taken to determine, once and for all, whether it is innocent or organic; should it prove innocent, a negative diagnosis of "No heart disease" is not enough; a positive diagnosis of "A healthy heart" is required, and thereafter the murmur should be ignored.

Treatment.—Early diagnosis is of the utmost importance *for treatment*; the longer the idea of heart disease or heart inferiority has been present, the more difficult it becomes to eradicate; and in the course of time it may become permanently fixed. In many cases the diagnosis is clear beyond all doubt when the patient is first seen, and in these circumstances elaborate investigation does more harm than good. Should there be any doubt about the diagnosis, immediate steps should be taken to confirm or refute it, and pending the result the physician's attitude should be absolutely non-committal.

Having *once confirmed* the diagnosis, the patient must not only be told that he has a healthy heart, but must be induced to believe this. He should be told that he is temporarily out of training as a result of insufficient sleep or of nervousness, and that he will make a complete recovery as a result of his own efforts. If there is insomnia or much tremor, sedatives should be ordered; the bromide and ergot mixture recommended for precordial pain of nervous origin (p. 435) is effective. A course of graduated outdoor exercise should be prescribed and should be insisted upon, some patients are inclined to slack or to funk the exercise unless they are under supervision. At an early stage the exercise should have reference to something useful (work) or to something interesting (games). This is the basis of "occupational therapy" as used in institutions, but with a little ingenuity it can be applied in the patient's own home An early return to work should always be advocated; prolonged periods on the sick list are demoralising.

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CHAPTER 21

HEART DISEASE IN RELATION TO PREGNANCY OR SURGICAL OPERATIONS

Pregnancy and Heart Disease

In the healthy woman, the advanced stage of pregnancy may give rise to certain cardiovascular symptoms and signs. The load on the heart reaches its maximum at the 30th week, then diminishes during the last four weeks. There is frequently a certain degree of breathlessness on exertion associated with the abdominal enlargement and the impeded descent of the diaphragm. There may be oedema of the ankles (often associated with varicosity of the leg veins) due to pressure of the uterus. Attacks of faintness associated with a fall in blood pressure are not uncommon The raising of the diaphragm tends to displace the cardiac apex upwards and outwards, and to give rise to changes in the electrocardiogram which have been described under the heading of "transverse heart electrocardiograms" (p. 163). *These symptoms and signs must not be regarded as indicating heart disease*, nor do they justify any attempt to "treat" the alleged "cardiac weakness" which does not in fact exist Finally, pregnancy is often complicated by anaemia which in itself may give rise to cardiovascular symptoms and signs, treatment is that of the anaemia

When a patient with organic heart disease becomes pregnant and reaches the advanced stage, her exercise tolerance is reduced and her cardiac symptoms become aggravated by the same factors. Thus a patient whose activities have been mildly limited before pregnancy tends to become more seriously limited; one whose limitations have been moderate may develop established failure These facts are important in attempting to assess the risk of pregnancy in a cardiac patient. Additional factors which require consideration include the risk of pregnancy toxaemia with hypertension, and of puerperal sepsis which, if it does not prove immediately fatal, may be followed by bacterial endocarditis On the opposite side of the scale must be weighed the psychological effects of sterility

if pregnancy be forbidden, and the risk attached to termination of pregnancy if this is already present.

The risks attaching to pregnancy in cardiac patients have been extensively studied by Bramwell and Longson, to whose writings the reader is referred for more detailed information. Here I propose only to give the main criteria on which a decision to allow or to forbid pregnancy should be based. The first, and most important, consideration is the patient's exercise tolerance prior to the pregnancy. If compensation has been adequate and the patient has never had symptoms of cardiac insufficiency, being able to undertake her household duties without discomfort, the risk is slight, the death rate in Bramwell's series was 4·9 per cent in primigravidae and 1·9 in multiparae When limitation was present prior to the pregnancy, the death rate rose to 10 per cent in primigravidae and 6 4 per cent in multiparae A history of repeated or persistent cardiac failure is an absolute bar to pregnancy. Other factors requiring consideration are age (the risk being considerably greater above the age of 30 in multiparae, but equal in primigravidae), social conditions, and the possibility of adequate antenatal supervision. The importance of antenatal supervision is shown by the fact that the death rate in cases admitted to hospital as "urgencies" was 44 per cent as compared with 1 per cent in the "quiet cases" It has recently been increasingly recognised by cardiologists that valvular lesions are not a bar to pregnancy provided the remaining indications are favourable Bramwell takes a similar view in the case of auricular fibrillation and of cardiac enlargement, neither of which he considers an absolute bar if compensation has been adequate previous to the pregnancy.

When pregnancy is already established in a case which is considered unsuitable it should be terminated within the first three months it must be remembered that the procedure is not entirely devoid of risk If the third month has been passed it is safer to allow the pregnancy to continue to term and to undergo natural delivery Opinion in recent years has been gradually changing in favour of natural delivery rather than Caesarian section or therapeutic induction, of the three methods, natural delivery has the lowest mortality in cardiac patients Patients with no history of previous failure should be kept under observation with a view to detecting the earliest

signs of failure Gilchrist recommends *that patients with a* history of previous failure should be confined strictly to bed for the remainder of their pregnancy even though no signs of failure are apparent; in his series the incidence of failure was reduced from 22 per cent to 4 per cent by this procedure. Failure is treated by rest and digitalisation, with confinement to bed for the remainder of the pregnancy; under no circumstances *should any interference be undertaken while failure is present*; and when the failure has been successfully treated, the urge to interfere should be resisted. In all cases the general health should be treated and, in particular, anaemia should be corrected.

Heart Disease and Surgical Operations

The physician is often asked by his surgical colleague to assess the risk of operation in a patient with organic heart disease. In coming to a decision on this point two factors must be taken into account, namely, the presence or absence of cardiac failure and the nature of the operation.

Many patients with heart disease stand operation remarkably well, comparing very favourably with those whose hearts are healthy. Those whose exercise tolerance has been good, whose pulse rates are slow, and who show no signs of cardiac failure are good operation risks, whether their cardiac lesion be valvular, hypertensive, or coronary; those who have had slight or moderate limitation are reasonable risks provided time is allowable for a preliminary period of rest for a few days. A history of recent cardiac failure greatly increases the danger of operation, as does the presence of failure, except in extreme emergency, it is safer to adopt expectant treatment in these cases and to allow adequate time for treatment of the heart failure. Patients with active inflammatory lesions, and those who have had a coronary thrombosis within the previous three to four weeks, are in grave danger from operation. Tachycardia is an unfavourable sign, whether it be due to the cardiac lesion or to a superimposed neurocirculatory asthenia

As regards the operation, the main guide is the amount of shock likely to ensue. The degree of shock depends to some extent on the nature of the operation, and a simple operation should always be preferred to a more elaborate one. Yet the

degree of shock can be minimised to a considerable extent if time is available for adequate pre-medication. Few conditions require immediate operation, incapable of being postponed for a day or two, and such postponement may make all the difference in a patient with heart disease. Even in cases of gastric or duodenal perforation it is safer to allow an hour or two for combating shock than to operate immediately without pre-medication

In the event of the appearance of cardiac symptoms following operation in any patient, the diagnostic criteria in no wise differ from those applicable in the absence of operation. There are two points, however, which should be borne in mind. Firstly, pulmonary embolism may simulate coronary thrombosis; of the two, pulmonary embolism is much more likely as a sequel to operation. Secondly, the development of tachycardia with a systolic murmur after operation is more likely to be due to sepsis than to endocarditis

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SECTION 4

CHAPTER 22

THE TREATMENT OF CARDIAC FAILURE

THE preceding chapters have covered the aetiology, symptoms and signs, and prognosis of the many conditions which may lead to cardiac failure. Many aspects of its treatment have already been discussed in dealing with these individual diseases. Here I propose to deal with those aspects of treatment which are of general application. What follows applies in the main to both left and right ventricular failure; where any differences are applicable they are noted.

Treatment will be discussed under the following main headings:

(1) Rest, nursing, and diet.
(2) Sedatives and the relief of distress or insomnia
(3) Digitalis and other cardiac tonics
(4) Diuretics.
(5) Mechanical measures for the relief of oedema
(6) Convalescence and rehabilitation

Rest and Nursing.—While slight degrees of impairment of cardiac reserve are treated by suitable limitation of activities, complete rest in bed is required for established failure or for severe impairment of reserve. Nursing is of the utmost importance. Patients with pulmonary congestion are propped up with pillows or a bed rest, a pillow below the knees prevents slipping down in bed If orthopnoea is severe they may lean over a bed table or sit in an arm-chair special cardiac beds permit a sitting posture Lowering the legs lessens pulmonary congestion and eases distress, though oedema of the feet is increased Cases with shock are nursed flat or with the foot of the bed raised. If there is neither pulmonary congestion nor shock the patient may choose his own posture If a bedpan causes straining, he may be lifted on to a commode Pressure points should be treated regularly and in chronic cases an air ring should be used; patients should be moved occasionally so that pressure is not constantly applied to the same parts.

The diet should be light and nutritious. Where there is much vomiting or severe respiratory distress it will consist mainly of milk, milk foods, custards, jellies, glucose drinks, and the like. Where there is no vomiting, ordinary light diet may be given, avoiding only heavy or greasy foods. In the convalescent stages dietetic restrictions are not required, save the advice that meals should be small; it is better to give an extra meal in the day than a large bulky meal. When there is oedema, salt and fluid are restricted. As regards salt it is usually sufficient to forbid the use of additional salt at meals, salt being used in the ordinary way in cooking; but in cases of refractory oedema it may be necessary to restrict the use of salt in cooking. As regards fluids, the allowance should not exceed two pints per day including tea, milk, soup, or other fluid taken with meals; many physicians allow only 1½ pints daily, but some patients suffer from severe thirst with this allowance. The fluid intake and the urinary output should be measured and recorded along with a four-hourly pulse and temperature chart. The bowels may be opened initially with a dose of calomel or castor oil, and should subsequently be kept acting by enemata or by gentle laxatives; small doses of cascara given three times a day are usually effective.

In most cases the measures outlined above, when combined with digitalisation and use of diuretics, will result in disappearance of oedema. A few cases prove refractory; in some others oedema returns soon after the patient is allowed up, when he becomes ambulant, or when he returns to work. In many of these a low sodium diet will abolish or control oedema and breathlessness; it will also help some of those with left heart failure in whom pulmonary oedema or cardiac asthma keep recurring while confined to bed or on resumption of activity. Special sodium free bread is required, and articles made with baking powder (scones, cakes, etc.) are prohibited; biscuits baked without baking powder or bread made with yeast may be substituted; bicarbonate of potash may be used as a "raising agent", the quantity required being half the amount of baking soda ordinarily used; no salt is allowed either in cooking or at meals; salted butter, meat, and fish are prohibited; and drugs containing sodium (e.g. "diuretin") are withheld. Most of the proprietary salt substitutes contain sodium salts other than the chloride and they cannot be used with this diet; a mixture

LOW SODIUM DIET

Containing approximately 0·8 gm Sodium (2 gms. NaCl) per day

BREAKFAST: Porridge with milk from ration, or cream.
" Salt-free " bread with " Salt-free " butter from ration.
Marmalade or jam
Tea with sugar, if desired, and milk from ration.
Egg or white fish or meat.

FORENOON: Fruit juice.

DINNER: No soups.
Serving of meat, white fish, tripe, rabbit or chicken
Serving of fresh vegetables, raw or cooked, without salt.
Potatoes.
Milk pudding made with milk from ration
Portion of fruit—fresh or stewed. Cream if desired.

AFTERNOON. " Salt-free " bread and butter or margarine
Jam
Tea, as *before*.

TEA: Serving of white fish, meat or egg.
Salad, if desired.
" Salt-free " bread and butter or margarine.
Jam
Tea, as before

SUPPER: " Salt-free " bread and butter or margarine.
Jam or syrup or honey.
Fruit juice or tea

NOTES.—*Daily rations* ½ pint milk. 1 oz salt-free butter or margarine
No cheese, bacon, smoked fish, tinned meats or canned vegetables to be taken
Most prepared breakfast cereals contain salt and are not permissible
No salt to be used in cooking or added to food
No baking soda, baking powder, or self-raising flour to be used.
" Salt-free " bread (baked with yeast) must be specially ordered from a firm able and willing to supply it
" Salt-free " butter and margarine can be obtained as ration from usual dealer
Neo Selarom may be used as a " salt substitute ".

This diet may be adapted for weight reducing in the case of obese patients, by omitting sugar, marmalade, jam, honey, and cream, and specifying maximum quantities of bread, oatmeal (as porridge), and potatoes so as to yield 1600 calories

of powdered potassium chloride and starch (1 part to 3) is recommended instead. (See also p. 458.)

With this regimen it is essential to maintain the patient's blood chloride level; for this purpose 30 to 60 grains of ammonium chloride are given daily in divided doses (four to eight capsules each containing 7½ grains). Patients who cannot tolerate ammonium chloride may take dilute hydrochloric acid instead. In many cases mercurial diuretics are not required, but if they are given, the dose of ammonium chloride should be increased. Restriction of fluids is unnecessary and undesirable; in fact, results are often better if patients on a low sodium diet are encouraged to drink 3 or 4 pints of fluid daily. Inadequate dosage with ammonium chloride sometimes precipitates a fall in blood chloride with a marked drop in blood pressure, severe asthenia, and mental confusion; uraemia is frequent but not invariable. This syndrome is not common, but is usually fatal when it does develop; even though the blood chlorides are restored by hypertonic saline transfusion, the asthenia, hypotonia and mental changes seem to be irreversible. The blood electrolytes should therefore be estimated at least weekly for the first two or three weeks of treatment, and from time to time thereafter. With careful supervision the treatment can be continued indefinitely, and cases of relapsing cardiac failure can often be kept ambulant for long periods; cessation of treatment in this type of case is soon followed by return of oedema.

Sedatives.—Relief of pain or of severe respiratory distress is a matter of urgency; for this purpose morphine is the drug of choice in an adult, nepenthe in a child. For an adult, ¼ grain of morphine should be given and repeated as necessary; the dose of nepenthe for a child is 1 minim for each year of the child's age. As soon as pain or distress have been relieved, the morphine must be discontinued to avoid the development of a habit; I have known patients deliberately work themselves up into a state of distress in order to secure further injections of morphine; should this occur, the most effective method of dealing with it is to give an injection of sterile water. The paroxysms of nocturnal dyspnoea which form such a distressing feature of left ventricular failure are also best treated with morphine, and [illegible]

by giving a dos[illegible]

subsequently [illegible] [illegible] other sedative (for example,

LOW SODIUM DIET

Containing approximately 0·8 gm. Sodium (2 gms. NaCl) per day

BREAKFAST :	Porridge with milk from ration, or cream.
	"Salt-free" bread with "Salt-free" butter from ration
	Marmalade or jam.
	Tea with sugar, if desired, and milk from ration
	Egg or white fish or meat.
FORENOON :	Fruit juice.
DINNER :	No soups
	Serving of meat, white fish, tripe, rabbit or chicken.
	Serving of fresh vegetables, raw or cooked, without salt.
	Potatoes.
	Milk pudding made with milk from ration
	Portion of fruit—fresh or stewed. Cream if desired.
AFTERNOON :	"Salt-free" bread and butter or margarine
	Jam
	Tea, as before
TEA :	*Serving of white fish, meat or egg*
	Salad, if desired
	"Salt-free" bread and butter or margarine.
	Jam.
	Tea, as before
SUPPER :	"Salt-free" bread and butter or margarine
	Jam or syrup or honey
	Fruit juice or tea

NOTES.—*Daily rations* ½ pint milk 1 oz. salt free butter or margarine

No cheese, bacon, smoked fish, tinned meats or canned vegetables to be taken

Most prepared breakfast cereals contain salt and are not permissible

No salt to be used in cooking or added to food.

No baking soda, baking powder, or self-raising flour to be used

"Salt-free" bread (baked with yeast) must be specially ordered from a firm able and willing to supply it.

"Salt-free" butter and margarine can be obtained as ration from usual dealer

Neo-Selarom may be used as a "salt substitute"

This diet may be adapted for weight reducing in the case of obese patients, by omitting sugar, marmalade, jam, honey, and cream, and specifying maximum quantities of bread, oatmeal (as porridge), and potatoes so as to yield 1600 calories.

As the result of data obtained from auricular catheterisation, McMichael contends that the beneficial action of digitalis in heart failure is due to the fact that it lowers venous pressure; when the summit of Starling's curve has been passed and the heart is overloaded, the lowering of venous pressure results in an increased cardiac output, if the summit of Starling's curve has not been passed, digitalis reduces the cardiac ouput by lowering the venous pressure, and is not only valueless but actually harmful. While accepting that digitalis does in fact lower venous pressure and influence cardiac output in this way, many physicians still hold the view that it also has a direct action on the myocardium, and that the fall in venous pressure is in part due to improvement in the cardiac tone and cardiac output. They also continue to regard the slowing effect of digitalis in auricular fibrillation (p. 138) as of value in relieving congestive failure associated with this condition.

Digitalis is indicated both in right and in left ventricular failure. It is more effective and more dramatic in right than in left ventricular failure, but is also of benefit in many cases of the latter. It is more effective, as a rule, in failure secondary to rheumatic heart disease than in the other varieties, as many cases of rheumatic heart disease are associated with auricular fibrillation. digitalis is more effective when there is auricular fibrillation than with a normal rhythm. If cases of rheumatic heart disease are excluded from consideration, the presence or absence of auricular fibrillation appears to make little difference to its efficiency. The question of its use in emphysematous heart failure has been discussed on p. 355. I must insist that the indication for digitalis is *heart failure*—not heart disease, not auricular fibrillation, not tachycardia, but heart failure alone. Nor is digitalis of any value in peripheral circulatory failure. It should be pointed out that in the absence of heart failure, digitalis is either ineffective or harmful, and should on no account be given, it is especially likely to do harm in cases of active carditis or the early ...

... every four hours, and these doses should be continued until full digitalisation has been secured, this is shown by relief of respiratory distress, com-

20 grains of chloral hydrate) may be tried. The only contra-indication to the use of morphine in doses sufficient to relieve pain or distress is in patients with severe kyphoscoliosis; in them, morphine is likely to prove fatal. For insomnia, restlessness, or delirium which are not due to pain or respiratory distress, 20 grains of chloral hydrate, with or without 30 grains of potassium bromide, is an effective and safe sedative, alternatively 1 grain of phenobarbitone may be given. It is of importance to ensure that patients with cardiac failure obtain adequate sleep.

Digitalis.—The digitalis group of drugs includes digitalis purpurea, digitalis lanata, strophanthus, and squill; from each of these, various active principles have been isolated. The majority of physicians nowadays confine themselves to the use of one or other of the preparations of digitalis, strophanthin is occasionally used intravenously for urgent cases, but digoxin is equally effective. It is important to acquire experience of the effects of one preparation rather than to use half a dozen and gain a thorough knowledge of none. Powdered digitalis leaf and standardised tincture of digitalis are adequate for the vast majority of cases, there is little to choose between them except that alcohol is in short supply at present and the tincture is a little more expensive; as against this, a finer graduation in dosage is possible. The active principles are considerably more expensive, and except for urgent cases, for those with severe vomiting who require intravenous therapy, and for those with an idiosyncrasy to oral digitalis, they have few advantages, in such cases digoxin or strophanthin may be used. Digoxin is thought to be more rapidly excreted and therefore less liable to cumulative action. It is usually stated that ½ grain of powdered leaf is the equivalent of 5 minims of the tincture, but my own impression is that the powdered leaf is more nearly equal to 7½ minims of tincture in its therapeutic effect. Accepting the standard figure, equivalent doses of the different preparations are as follows

Digitalis leaf	gr. ½	gr. 1	gr 1½	grs 2	grs 2½	gr 3
Tr. Digitalis .	m 5	m. 10	m 15	m. 20	m 25	m 30
Digoxin .	—	—	¼ mg	—	—	½ mg.
Digitaline Nativelle .	—	$\frac{1}{600}$ gr.	—	—	$\frac{1}{240}$ gr.	—

($\frac{1}{600}$ grain = $\frac{1}{10}$ milligram, $\frac{1}{240}$ grain = ¼ milligram)

shown that it has a venous pressure-reducing effect similar to that of digitalis; its action on pulmonary congestion is more powerful than that of digitalis, but its action on systemic venous congestion is relatively less potent. It also appears to have an action on the coronary vessels producing vaso-dilatation. It gives good results in cases of left ventricular failure; the disappearance of Cheyne-Stokes breathing and cardiac asthma is often dramatic, and its effects in left heart failure are more striking than those of digitalis. In some cases a combination of theophylline-ethylene-diamine with digitalis produces a more satisfactory response than either drug alone. Theophylline-ethylene-diamine is also valuable in the treatment of paroxysmal anginal attacks. It may prove useful in those cases of congestive failure in which the pulse is slow and digitalis is considered undesirable. It can be given intravenously (0.24 to 0.48 gm.) or intramuscularly; oral tablets of 0.1 gm. four-hourly also give good results. Various other drugs have from time to time been recommended as coronary vaso-dilators, but controlled experiments have shown that they have little if any effect in human heart failure.

Diuretics.—*In many cases rest and digitalis alone produce a good diuresis, followed by disappearance of all oedema; in these circumstances diuretics are not required. In some cases digitalis relieves the dyspnoea and slows the pulse rate, but oedema persists. Less frequently digitalis has little or no effect until a diuretic is given as well. In either of the latter types of case diuretics should be used. They may be given either simultaneously with digitalis or independently; cases with considerable oedema and a slow pulse rate are often suitable for diuretics alone. The mercurial diuretics are also of considerable benefit in cases of left heart failure with pulmonary oedema; dramatic relief often occurs within a few hours of their administration. They are contra-indicated where there is evidence of renal damage (nephritis), but in all other cases they are the diuretic of choice. Acute retention sometimes follows their use in patients with prostatic hypertrophy.*

By far the most effective diuretics are the organic mercurial compounds, mersalyl, "neptal", "novurit", and "salyrgan". In efficacy, there is little to choose between them. For routine use they are best given intramuscularly. Many patients respond very well to intramuscular injection; a few respond

mencing diuresis, and a fall in pulse rate to the region of 80, and it usually requires the equivalent of some 5 drams of the tincture. In urgent cases, larger doses may be given at the start; or the total dose may be calculated as described on p. 139 and given in a single dose or in three divided doses. Once full digitalisation has been secured, the larger doses are stopped and the patient is put on a maintenance dose, this is usually from 20 to 30 minims of the tincture or from 2 to 3 grains of the powdered leaf daily. Patients vary considerably in their sensitivity to digitalis and in their rate of excreting the drug; some can tolerate as much as 45 minims of the tincture daily while others develop toxic symptoms with as little as 15 minims daily. The necessary dose for any given patient must be found by trial coupled with careful observation of the symptoms, the pulse rate, and the urinary output.

Signs of digitalis intoxication must always be carefully sought in patients who are under treatment with digitalis. The earlier signs are nausea, vomiting, dizziness, slowing of the pulse to below 60, or appearance of extrasystoles. A little later the pulse becomes coupled, the urinary output falls, oedema reappears or increases, and the tongue becomes furred. Slowing of the pulse is not always present; symptoms of intoxication sometimes appear while the pulse rate is still above 100. If these signs are overlooked and the digitalis is continued, paroxysms of tachycardia may appear, causing the pulse to be rapid and irregular once more. Not infrequently, digitalis poisoning gives rise to paroxysmal attacks of anginal pain. In the advanced stage of digitalis intoxication, the picture is difficult to distinguish from that of cardiac failure itself, there being a rapid irregular pulse, oedema, breathlessness, vomiting, oliguria, and perhaps attacks of anginal pain. Death from ventricular fibrillation may follow.

Some patients, having had the acute stage of their cardiac failure treated with digitalis, remain well without any maintenance dose, and in these cases no maintenance dose should be given. The indication for a maintenance dose is failure to maintain the improvement produced by the original full dosage.

Theophylline with Ethylene-Diamine.—This combination has become popular in recent years, it is marketed under various trade names including "cardophyllin", "deriphyllin", "euphyllin", and "genophyllin". Howarth and McMichael have

shown that it has a venous pressure-reducing effect similar to that of digitalis; its action on pulmonary congestion is more powerful than that of digitalis, but its action on systemic venous congestion is relatively less potent. It also appears to have an action on the coronary vessels producing vaso-dilatation It gives good results in cases of left ventricular failure; the disappearance of Cheyne-Stokes breathing and cardiac asthma is often dramatic, and its effects in left heart failure are more striking than those of digitalis. In some cases a combination of theophylline-ethylene-diamine with digitalis produces a more satisfactory response than either drug alone. Theophylline-ethylene-diamine is also valuable in the treatment of paroxysmal anginal attacks It may prove useful in those cases of congestive failure in which the pulse is slow and digitalis is considered undesirable It can be given intravenously (0·24 to 0·48 gm) or intramuscularly, oral tablets of 0·1 gm four-hourly also give good results Various other drugs have from time to time been recommended as coronary vaso dilators, but controlled experiments have shown that they have little if any effect in human heart failure

Diuretics.—In many cases rest and digitalis alone produce a good diuresis, followed by disappearance of all oedema, in these circumstances diuretics are not required. In some cases digitalis relieves the dyspnoea and slows the pulse rate, but oedema *persists* Less frequently, digitalis has little or no effect until a diuretic is given as well In either of the latter types of case diuretics should be used They may be given either simultaneously with digitalis or independently, cases with considerable oedema and a slow pulse rate are often suitable for diuretics alone The mercurial diuretics are also of *considerable benefit* in cases of left heart failure with pulmonary *oedema* dramatic relief often occurs within a few hours of their administration They are *contra*-indicated where there is evidence of renal damage (nephritis), but in all other cases they are the diuretic of choice Acute retention sometimes follows their use in patients with prostatic hypertrophy

By far the most effective diuretics are the organic mercurial compounds, mersalyl, "neptal", "novurit", and "salyrgan" In efficacy, there is little to choose between them. For routine [illegible]

res[illegible] Many patients

[illegible], a few respond

mencing diuresis, and a fall in pulse rate to the region of 80: and it usually requires the equivalent of some 5 drams of the tincture. In urgent cases, larger doses may be given at the start; or the total dose may be calculated as described on p. 139 and given in a single dose or in three divided doses. Once full digitalisation has been secured, the larger doses are stopped and the patient is put on a maintenance dose; this is usually from 20 to 30 minims of the tincture or from 2 to 3 grains of the powdered leaf daily. Patients vary considerably in their sensitivity to digitalis and in their rate of excreting the drug, some can tolerate as much as 45 minims of the tincture daily while others develop toxic symptoms with as little as 15 minims daily. The necessary dose for any given patient must be found by trial coupled with careful observation of the symptoms, the pulse rate, and the urinary output.

Signs of digitalis intoxication must always be carefully sought in patients who are under treatment with digitalis. The earlier signs are nausea, vomiting, dizziness, slowing of the pulse to below 60, or appearance of extrasystoles. A little later the pulse becomes coupled, the urinary output falls, oedema reappears or increases, and the tongue becomes furred. Slowing of the pulse is not always present, symptoms of intoxication sometimes appear while the pulse rate is still above 100. If these signs are overlooked and the digitalis is continued, paroxysms of tachycardia may appear, causing the pulse to be rapid and irregular once more. Not infrequently, digitalis poisoning gives rise to paroxysmal attacks of anginal pain. In the advanced stage of digitalis intoxication, the picture is difficult to distinguish from that of cardiac failure itself, there being a rapid irregular pulse, oedema, breathlessness, vomiting, oliguria, and perhaps attacks of anginal pain. Death from ventricular fibrillation may follow.

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better when the drug is given intravenously. The latter is a more dangerous method, in rare instances a fatal reaction has followed intravenous injection. The solutions are highly irritating to subcutaneous tissue, capable of causing necrosis or severe inflammation and pain; great care must be taken to avoid leakage during or after intravenous injection. A third route is by rectum in the form of a suppository; this method is sometimes effective, but often disappointing. Tablets of mersalyl and "neptal" are also available for oral administration; in some patients they give a very good result, but more often oral administration is ineffective. Whichever route is chosen, the urine should first be rendered acid by the administration of ammonium chloride; it is usually suggested that the patient should be given 15 to 25 grains of ammonium chloride thrice daily; I have had equally good results by giving a single dose of 30 grains in capsules 30 minutes before the injection, and I never use any other method now. As a routine, the drug is given every other day or every third day until the oedema has largely disappeared, then once or twice a week until it has entirely vanished. On occasion I have given the mercurial daily; it is necessary to be on the look-out for symptoms of mercurial poisoning.

Prior to the introduction of the mercurial diuretics, "blue pill" was popularly employed for this purpose. "Guy's pill" ("Bailey's pill") consists of 1 grain each of blue pill, digitalis leaf, and powdered squill. Other diuretics sometimes give a good result, but they are less powerful and less reliable than the mercurials. Theobromine or its compound "diuretin" (theobromine with sodium salicylate) is one of the best, the dose being 15 grains thrice daily. Theocin sodium acetate gave good results. Potassium citrate is rarely of value in cases which have failed to respond to digitalis alone.

Mechanical Measures for the Relief of Oedema.—Occasionally, despite the foregoing treatment, oedema persists and becomes so extreme that it threatens to rupture the skin unless relieved. In these circumstances acupuncture should be performed with particularly strict aseptic precautions. Acupuncture has entirely superseded the older method of drainage by Southey's tubes; the latter were apt to become blocked and were more difficult to keep aseptic; their only advantage was that the amount of drainage could be measured.

40 mg. per 100 ml in eight days while the blood sodium level fell by 20 mg per 100 ml. and oedema was only slightly reduced · in other cases, the blood potassium has remained stationary, serum sodium has fallen, and oedema has disappeared. Dosage for an average case is 15 grams in a glass of fruit juice or milk thrice daily

There is one important difference between a low sodium diet and an ordinary diet plus resodec Much of the sodium in an ordinary diet is combined with chloride, so that a low sodium diet is deficient in chloride also, additional chloride must be supplied Even with this safeguard, if the patient's renal threshold for chloride is low, there is a danger of *hypochloraemia* with symptoms resembling an Addisonian crisis On the other hand, with an ordinary diet and resodec, sodium is absorbed by the resin while chloride is absorbed by the patient; if his ability to excrete chloride is normal, no harm results, but if his kidneys have difficulty in excreting chloride (a condition usually associated with impaired *nitrogen excretion*) a state of *hyperchloraemia with acidosis and tissue dehydration* arises, this may be accompanied by uraemia In many cases the two methods are interchangeable, but some patients who do well on a low sodium diet cannot tolerate the resin, and, conversely, some who are in danger on a low sodium diet can safely be treated with the resin The capacity of the kidney to retain or to excrete chloride is the deciding factor.

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ment, but continue to recur as soon as the patient is allowed a little latitude. In such cases the operation of total thyroidectomy has been advocated. *It should be reserved for patients who have been given an adequate trial of medical treatment, despite which they remain, for all practical purposes, totally incapacitated or bedridden.* In suitably selected cases good results are obtained; the procedure lowers the basal metabolic rate and lessens the work of the heart. *Myxoedema may follow* the operation necessitating the use of thyroid, the optimum dose of which must be found by trial. In recent years attempts have been made to achieve the same result by giving thiouracil; while some successes have been claimed, I have so far been unable either to produce myxoedema or to influence chronic cardiac failure with thiouracil in doses up to 1 gm. daily.

The onset of right heart failure in a case of primary left heart failure is usually accompanied by relief of the more distressing symptoms. This fact has led Cossio to treat cases of intractable left heart failure by deliberately inducing right heart failure; a specially devised instrument is introduced into the jugular vein, passed through the superior vena cava and right auricle into the right ventricle, in its withdrawal the tricuspid valve is cut. Four out of five patients so treated by him experienced great relief, the fifth died

Sodium Absorbing Resins.—Attempts are now being made to secure the therapeutic advantages of a low sodium diet without the tastelessness and domestic difficulties involved in its use. For this purpose cation-absorbing resins are given along with ordinary diet These resins have been used in various industrial processes for a number of years Those employed medicinally absorb cations in an alkaline medium and release them in an acid medium *Resodec* is a mixture of one such resin with its potassium compound; it is given with ordinary diet and additional calcium either as milk or as calcium gluconate. Potassium is liberated in the stomach, sodium, potassium and calcium are absorbed in the intestine The net effect is (or should be, if dosage is correct) extraction of sodium from the diet without depletion of potassium or calcium. The method is still experimental and requires strict biochemical control with frequent estimations of the blood electrolytes, at any rate in the early weeks of treatment; in a case of the author's, the blood potassium rose from 20 to

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